

CARDIAC EMERGENCIES AND HEART FAILURE

Prevention and Treatment

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Illustrated



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PREFACE TO THE SECOND EDITION

DURING the past three years numerous advances have been made in cardiology and a number of new drugs have been introduced for the treatment of cardiac emergencies. In presenting this revision of the original text, those newer therapies have been stressed which, in our opinion, have proven successful and will continue to be utilized, procedures that appear promising but which have not yet stood the test of time are discussed only briefly. The chapters on congestive failure, arrhythmias, acute coronary disease, rheumatic heart disease and hypertensive crises have been enlarged or revised and case reports added to include newer concepts and to illustrate the use of drugs that have been recently introduced. The bibliography has been brought up to date and the index enlarged. Several charts and tables have been added in the section on arrhythmias. The book has been kept at "pocket size" so that the physician may have it available for use at all times, even at the bedside. As in the first edition, cases have been added from the authors' private practices and from the wards of the Mount Sinai Hospital. We are most grateful to our wives who gave us unceasing assistance in the preparation of this book.

New York, N.Y.

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PREFACE TO THE FIRST EDITION

THE prompt diagnosis of cardiovascular emergencies and their proper treatment are among the most common problems confronting the general practitioner. This monograph has been written in the hope that it will be of value to him as he encounters these acute conditions. It is not intended to be a book on Cardiology, Radiology or Electrocardiography, consequently, it does not cover these fields as thoroughly as do the standard textbooks. Physiology and pathology are discussed when a fuller understanding of them is considered necessary for the intelligent handling of cardiac patients. The drugs that have been found valuable are stressed, other drugs in current use are discussed. Case histories are presented to illustrate specific conditions and the use of the newer drugs. Throughout the book, the importance of preventing cardiac emergencies by adequate interval therapy is emphasized.

We have included sections on surgical cardiac emergencies, on cardiac resuscitation and on certain acute conditions that are not commonly encountered, *i.e.*, dissecting aneurysm, hypertensive crises secondary to pheochromocytoma, and the acute episode resulting from a "ball valve" thrombus, in the hope that when these emergencies are seen they may be recognized and treated properly.

The section on congestive heart failure and its complications is brief, but we have included all of the information

that we believe is necessary for the treatment of this condition

We have surveyed the literature, but have drawn freely from our own cases and experience, as well as from material previously published by one of us (A M M). Portions of this monograph have been presented in several broadcasts sponsored by the New York Academy of Medicine over Station WNYC (FM).

We wish to thank Dr. Samuel Kahn and Dr Phillip Samet for editorial assistance. We are indebted to members of the cardiographic department of the Mount Sinai Hospital for valuable suggestions.

We hope that the great number of physicians who are called upon to diagnose and treat the diseases that we discuss will benefit from this work.

New York, N Y

A M M
M M
H L. J

CONTENTS

CHAPTER	PAGE
1. Arrhythmias	
Paroxysmal Tachycardia	11
Supraventricular Tachycardias	12
Atrial Fibrillation	14
Quinidine	18
Digitalis	22
Atrial Flutter	31
Atrial and Nodal Tachycardia	35
Ventricular Tachycardia	42
Ventricular Fibrillation	47
Heart Block	49
Other Arrhythmias	51
2 Acute Pulmonary Edema and Congestive Heart Failure	54
Pathogenesis of Cardiac Failure	54
Paroxysmal Nocturnal Dyspnea	57
Acute Pulmonary Edema	58
Acute Cardio-pulmonary Insufficiency	62
Therapy of Congestive Heart Failure	64
3 Angina Pectoris, Coronary Insufficiency and Coronary Occlusion	76

CHAPTER	PAGE
Treatment	76
<i>Angina Pectoris</i>	77
<i>Status Anginosus and Angina Decubitus</i>	80
<i>Coronary Insufficiency</i>	83
<i>Spontaneous Coronary Insufficiency</i>	85
<i>Coronary Insufficiency Secondary to Other Conditions</i>	88
<i>Coronary Thrombosis or Occlusion</i>	89
<i>Anticoagulant Therapy</i>	101
<i>Pulmonary Embolism</i>	104
<i>Other Oral Anticoagulants</i>	108
4 Syncope	113
5 Rheumatic Fever and Heart Disease	123
<i>Congestive Failure</i>	124
<i>Acute Pericarditis</i>	126
6 Hypertensive Encephalopathy and Crises— <i>Pheochromocytoma</i>	132
<i>Pheochromocytoma</i>	136
7 Dissecting Aneurysm	139
8 Traumatic Heart Disease	142
9 Surgical Emergencies in Cardiovascular Dis- <i>ease Their Prevention and Treatment</i>	147
<i>Surgery in the Cardiac Patient</i>	147
<i>Cardiac Arrest</i>	151

LIST OF ILLUSTRATIONS

FIGURE	PAGE
1 Effect of Potassium in Digitalis Toxicity	27
2 Auricular Flutter. Effect of Intramuscular Quinidine	32
3 2.1 Auricular Tachycardia	34
4 Nodal Tachycardia. Effect of Pronestyl	37
5. Ventricular Tachycardia. Effect of Pronestyl	44
6 Complete A-V Block and Ventricular Fibrillation	48
7 Acute Coronary Insufficiency Following Exertion	84
8 Acute Coronary Insufficiency Caused by Hemorrhage	87
9 Electrocardiographic Changes of Coronary Insufficiency During the Premonitory Phase of Coronary Occlusion with Anterior Infarction	90
10. Acute Coronary Occlusion with Posterior Infarction	91
11. Acute Coronary Occlusion with Diaphragmatic Infarction	100
12 Pulmonary Embolism with Coronary Insufficiency	103
13. Effect of Carotid Sinus Pressure	116
14. Acute Pericarditis	125
(8)	

Cardiac Emergencies and Heart Failure

INTRODUCTION

As a result of the tremendous advances in the prevention and treatment of many diseases, and of the increased span of life, cardiovascular disease now greatly outnumbers all others and causes more than half the deaths after the age of fifty. Acute heart conditions require emergency treatment more than do those of any other organ, making the term "acute heart" even more appropriate than the expression "acute abdomen." The prompt treatment of acute cardiac disease by the application of well-established rules, and the utilization of recent advances in the field of cardiology will save many lives and hasten recovery in others. Since treatment must be immediate, it is incumbent upon the physician to know how to diagnose and treat cardiac emergencies, and to have the essential drugs readily available. This requires the careful selection and constant replenishment of medical supplies, for it is very disheartening to make the correct diagnosis of a cardiac emergency, and not to have the suitable medication available. The following material should always be present in the doctor's bag in adequate amount. Additional drugs, such as intravenous hexamethonium (Chapter 6), which are generally reserved for hospital use, need not be carried but should be available in a hospital.

1. Aminophyllin, ampules, 10 cc. = 24 gm for i v use,
2 cc = .48 gm for i m. use.

- 2 Atropine Sulfate, 75 mg. (gr. 1/100) hypodermic tablets for i v. use
- 3 Cedilanid (Lanatoside C), 4 cc. (.8 mg) and 2 cc. (4 mg) ampules for i v or i m. use.
- 4 Demerol, 2 cc. (100 mg) ampules, or multiple dose vials (1 cc = 50 mg.)
- 5 Digoxin, 25 mg tablets or Gitalgin, 5 mg tablets
- 6 Epinephrine 1:1000 (1 cc ampules)
- 7 Isuprel, 10 mg linguets
- 8 Mercurial Diuretic (Mercurhydrin or Thumerin) 1 or 2 cc.
- 9 Morphine Sulfate, 16 mg ($\frac{1}{4}$ gr) tablets for i m or i v use
10. Needles for phlebotomy
11. Neosynephrin, 1 cc = 10 mg , for i v or i m. use
- 12 Nitroglycerin hypodermic tablets, gr. 1/200 or gr. 1/150
- 13 Pronestyl (Procaine Amide), 10 cc ampules (1 cc. = 100 mg) and 250 mg tablets.
- 14 Quinidine, 1 cc ampule = 2 gm. for i m use, 2 gm (3 gr) tablets
- 15 Sterile water for dilution of tablets.
- 16 Ouabain (C Strophanthin) 2 cc. ampules = 5 mg , or Strophanthin K ("Strophosid"), 1 cc ampule = 5 mg
- 17 Thorazine, 1 cc = 25 mg for i m use, 10 or 25 mg tablets or Dramamine, 50 mg. tablets, 10 cc ampules (1 cc = 50 mg)
- 18 Tourniquets
- 19 Vasovyl, 1 cc = 25 mg , or Wyamine, 1 cc = 15 mg for i v. or i m. use

Chapter

1

ARRHYTHMIAS

A CHANGE in the rate or rhythm of the heart is a common cause of acute distress in patients with and without cardiac disease. Even in a patient with a normal heart, an arrhythmia may produce symptoms of anxiety, precordial aching or severe pain,¹¹ vertigo, syncope, vomiting, collapse, and rarely congestive heart failure.¹² Severe circulatory derangements occur following the onset of arrhythmias with a rapid rate, *i.e.*, the paroxysmal tachycardias, or a slow rate, *i.e.*, complete A-V block. This is especially true in elderly people.

PAROXYSMAL TACHYCARDIA

The paroxysmal tachycardias include atrial flutter and fibrillation, atrial, nodal, and ventricular tachycardia and ventricular fibrillation. (The term "atrial" is to be preferred to "auricular" since the cardiac pacemakers are located in the atrium and not in the appendage (auricle).) Any of the tachycardias may remit spontaneously within several hours and cause no disturbance, but they may produce severe symptoms and marked effects on circulatory dynamics. The clinical severity of any arrhythmia is usually proportional to the increase in the ventricular rate. It has been demonstrated that a change in heart rate from 70 or 80 to one of 120 or 130 usually increases minute volume output. This occurs despite the fact that ventricular filling and stroke volume may be decreased. (The cardiac output,

which is the product of heart rate and stroke output, actually increases.) With further increases in cardiac rate, total minute volume is reduced.¹²⁵ This may result in severe cerebral anoxia with fainting or convulsions. For example, J.C., a thirty-two-year-old factory manager studied by us, fainted on several occasions. He gave a long history of discomfort whenever he wore a tie, and his pulse slowed moderately on carotid sinus pressure. The fainting was, therefore, considered to be the result of bradycardia caused by carotid sinus hypersensitivity. Close questioning, however, revealed that the syncope was preceded by palpitations, and he was later seen during an episode of nodal tachycardia. This was probably the major cause of his fainting although the carotid sinus may also have played a role.

If tachycardia persists, it may result in a further decrease in cardiac output and the clinical picture of shock, cardiac failure,¹⁰⁶ and/or coronary insufficiency with chest pain and RS-T and T-wave changes in the electrocardiogram. A patient demonstrating this syndrome was a sixty-five-year-old woman with carcinoma of the left lung and hemothorax who was receiving nitrogen mustard therapy. On the fifth day after therapy she suddenly experienced precordial pain and palpitation and went into shock. Her heart rate was 160 and an electrocardiogram revealed a 2:1 atrial flutter with RS-T depressions in all leads. While the cardiogram was being taken, the rhythm reverted to normal sinus rhythm but the RS-T depressions persisted for several minutes. In some instances RS-T depressions persist for hours or days.

THE SUPRAVENTRICULAR TACHYCARDIAS

Supraventricular tachycardias, i.e., atrial or nodal tachycardia and atrial flutter and fibrillation, occur in persons with normal or abnormal hearts,^{42, 112} and may be precipi-

tated by emotional disturbances.³⁷⁹ Occasionally, they are the only indication of hyperthyroidism and this disease should be ruled out in all cases of recurrent paroxysmal tachycardia. Although the basal metabolic rate is within normal limits in approximately 85 per cent of patients with supraventricular tachycardia,¹⁷⁵ other tests of thyroid function (radioactive iodine [I_{131}] uptake, and blood protein bound iodine) may indicate a hyperthyroid state. *Ventricular tachycardia*, on the other hand, is almost always associated with organic heart disease, particularly coronary occlusion,¹⁸⁶ and is of more serious import.

In many normal persons with paroxysmal tachycardia therapy may be withheld for several hours since these arrhythmias often remit spontaneously. People with normal hearts may be able to tolerate rapid heart rates for weeks or months without exhibiting any detectable alterations in circulatory function.³⁸⁰⁻³⁸² Some patients, however, become emotionally disturbed and may experience precordial discomfort as a result of rapid beating of the heart, in such patients, especially those with organic heart disease, treatment should be started immediately. Similarly, therapy of the specific tachycardia should not be delayed, even in normal persons, if the patient cannot be observed periodically.

Some general measures are of value in the prevention and treatment of all tachycardias, namely, rest, sedation, and the avoidance of tobacco, alcohol, caffeine, and emotional upsets. For example, a twenty-eight-year-old physician, recently observed, developed atrial fibrillation whenever he took one drink and smoked one cigarette simultaneously. He was finally persuaded to stop drinking and smoking and experienced no further attacks. Eating fatty foods or large meals on rare occasions produces atrial arrhythmias in patients with otherwise normal hearts. We have observed a patient who suffered a bout of atrial tachycardia each time he ate a frankfurter or a large dish of ice cream. The

avoidance of these foods completely eliminated the attacks.

ATRIAL FIBRILLATION

Atrial fibrillation is the most common paroxysmal arrhythmia and occurs most frequently in patients with rheumatic and arteriosclerotic heart disease^{11 123} but both the paroxysmal and chronic types are observed in the absence of organic heart disease.^{213 232} It may be seen in cases of Graves' disease,²⁰ following operative procedures,¹⁶⁴ in pneumonia and in other febrile illnesses.

Atrial fibrillation has been attributed by some observers to rapid, irregular circus movements initiated in the atrial muscle and perpetuated by the occurrence of local blocks,¹³⁶ but this view has been challenged by many authors.^{112 171 231 231} These latter investigators believe that *atrial fibrillation* is a chaotic disturbance of atrial rhythm in response to stimuli originating in irritable foci in the atrial muscle and that there is no element of circus movement. According to this opinion, all atrial arrhythmias have a similar mechanism, the only difference between them being the number and frequency of impulses arising from the single focus.²⁶² This view has not as yet been universally accepted¹³⁷ but would appear from the evidence presented to be a logical explanation of atrial arrhythmias.

The paroxysmal form of atrial fibrillation usually sets in abruptly with a ventricular rate between 120 and 160 and a totally irregular rhythm (Table 1). A pulse deficit may be present. The rhythm may revert to normal with rest and the use of sedatives, but if fibrillation persists treatment with either quinidine or digitalis should be initiated, digitalis being given in all cases of atrial fibrillation with cardiac failure. Some observers favor the use of *digitalis*, particularly intravenously, even when congestive failure is not present. Although its primary effect in these cases is to slow the ventricular rate, sinus rhythm is occasionally in-

Table 1. Clinical Differentiation of Common Supraventricular Arrhythmias

TYPE OF CARDIAC ARRHYTHMIA	Rate/Min	Rhythm	Effect of Pressure on Pulse Rate (Range)	Effect of Carotid Sinus Pressure	Other Diagnostic Features
Normal Tachycardia	90-160	Regular	Increases rate	None or temporary slowing	<ol style="list-style-type: none"> Not over 160/min No sudden onset No real effect of carotid sinus pressure
Paroxysmal (premature contraction)	Usually normal	Randomly regular	May also slow the rate	No effect	<ol style="list-style-type: none"> Normal rate Precipitants are followed by long interval thus usual Recovery may include extrasystoles
Paroxysmal Atrial Tachycardia	160-250 (usually 180-200)	Regular	No effect	Returns to normal at rest	<ol style="list-style-type: none"> Rate over 160/min (usually 180-200) regular Sudden onset and equally sudden Almost returns to normal with carotid sinus pressure
Atrial Fibrillation	Ventricular Rate 120-150 Atrial Rate > 150	Totally irregular	Increases irregularity	No effect	<ol style="list-style-type: none"> Irregularity of rhythm with rapid rate Pulse deficit No effect of carotid sinus pressure Exaggerated only in cases of irregularity
Atrial Flutter	Ventricular Rate 125-160 (2:1 block) Atrial Rate 250-350	Usually regular, may be irregular	May double ventricular rate by temporarily abolishing 2:1 block	Temporary slowing	<ol style="list-style-type: none"> Rhythm regular or irregular Temporary slowing by carotid sinus pressure Flutter waves in neck

duced following digitalis administration. If quinidine is used (Table 2), 3 grains (2 gm) should be given orally every two hours. The first dose may be used to test sensitivity since reactions will usually occur within two hours if the patient is allergic to the drug¹⁰⁵ True sensitivity is unusual however, and treatment of a patient who is critically ill should not be delayed. If there is no change in

Table 2. Suggested Approach to the Therapy of Paroxysmal Atrial Fibrillation With Quinidine. No Cardiac Failure

1st Day

- 1 Rest and sedation
- 2 Quinidine : 0.2 gm every 2 hours for 2 doses, then
0.4 gm every 2 hours for 1 to 4 doses. No therapy at night*

If no response—2nd Day

- 3 Quinidine : 0.4 gm every two hours for 3 doses, then
0.6 gm every two hours for 3 or 4 doses.
If no response, quinidine usually will not be effective and patient should be digitized

*If patient has severe symptoms, quinidine should be continued during night in gradually increasing doses until 6 gm has been given for 3 doses.

rhythm after four hours, the dose is increased to 8 grains (4 gm) every two hours for three or four doses. Occasionally, it is necessary to raise the dose to 9 grains (6 gm) every two hours for three or four doses. Quinidine has relatively little cumulative effect and peak plasma levels are reached after 4 or 5 adequate doses have been given¹¹⁶ If the same dose is administered thereafter, the plasma level rises only slightly, and it is therefore necessary to increase the dose again to achieve an effect. If quinidine is initially administered every four to six hours peak levels may not be reached for from two to four days. The total daily or

weekly dose is of much less importance in determining plasma concentration than is the amount of the individual dose,^{210 729} as only traces of quinidine are found in the plasma twenty-four hours after the last dose has been given.

If regular rhythm is not restored with the dose of 9 grains, larger ones will rarely have the desired effect and the drug should be discontinued. Serious reactions (respiratory failure or asystole) may occur if quinidine is given in excessively large doses (over 2½ to 3 gms. in a twelve to twenty-four-hour period). Unfortunately there is little correlation between quinidine blood levels and the toxic effects of the drug on the myocardium, so that there is no way of accurately predicting the onset of "toxic effects." Excellent results are occasionally obtained with very small doses. It is often best to administer quinidine on a two-hour schedule for five or six doses during the day and omit night medication. In this way the patient can be more carefully observed. This, of course, should not be done when the patient has severe symptoms and a delay of several hours may be dangerous.

We have used quinidine *intramuscularly* during the past four years with good results. A suitable preparation is a 20 per cent solution of quinidine sulfate in propylene glycol, 3 grains (2 gm) per cc., although several other preparations are also available. The injection is rarely painful,¹¹³ the dosage is the same as when given orally, and toxicity is not increased when the drug is given by this route. Intramuscular medication is especially valuable when oral administration cannot be tolerated in patients who are acutely ill. Quinidine hydrochloride is also satisfactory for intramuscular use. Intravenous quinidine, which is now available as quinidine lactate, in 10 cc ampules, containing 65 gm., is dangerous and its use must be carefully controlled. There is little indication for the intravenous administration of quinidine as most cases in which quinidine will be effective respond to intramuscular or oral medication.

If fibrillation persists after a satisfactory trial with quinidine, the patient should be digitalized (*cule infra*). In all recalcitrant cases, it is essential to exclude the presence of hyperthyroidism or active rheumatic fever. In patients with atrial fibrillation and hyperthyroidism the prognosis is excellent, for, once the hyperthyroid state has been corrected,⁴ the arrhythmia will disappear in 95 per cent of these cases either spontaneously or following quinidine therapy.

If quinidine restores sinus rhythm, the dose is reduced gradually and a maintenance dose is instituted. Three to six grains (2 to 4 gm), three times a day may suffice but in occasional cases 6 to 9 grains (4 to 6 gm) four or more times a day may be required for long periods. When quinidine is given four times daily, the first dose is taken early in the morning on awakening, the second at midday, the third in the evening, and the fourth dose just before retiring, in order to maintain adequate blood levels. In unusual instances the last dose must be given during the night.

An example of this type of case is that of a fifty-nine-year-old white male, who had severe rheumatic heart disease and atrial fibrillation and was treated with 3 gm (5 gr) of quinidine sulfate orally every two hours, as outlined above. Regular rhythm was restored and he was maintained successfully on 3 gm , four times a day, awakening at 4 A M daily for the morning medication. Whenever this dose was omitted, fibrillation recurred.

Three-grain (2 gm) enemas of quinidine or quinidine in oil ("Quinidate") are available as "long acting" preparations to avoid giving a dose during the night. The drug in this form is more slowly absorbed, allowing for a greater interval between doses.

If attacks of fibrillation recur infrequently, quinidine need not be maintained more than a week after the acute episode. However, if paroxysms occur frequently, prophylactic quinidine should be continued indefinitely.

Occasionally, the attacks respond to quinidine with increasing difficulty as they become more frequent. In such cases it may be more advisable to slow the ventricular rate with digitalis than to attempt to restore sinus rhythm with quinidine. A good example of this therapy is presented by a woman of forty-four, who had had rheumatic heart disease with mitral stenosis for twenty years. She had developed a cerebral embolus three years ago and since then had experienced frequent atrial premature beats and episodes of atrial fibrillation. At first, these readily responded to quinidine but the attacks became very frequent and lasted several days in spite of large doses of this drug. The patient was greatly distressed during the attacks and developed great anxiety regarding their recurrence. In the next attack digitalis alone was given until the ventricular rate fell to 70. Since then, she has been maintained on 25 mg Digoxin daily, has remained in chronic atrial fibrillation but has been entirely comfortable.

Although recent studies have shown that adequate cardiac output can be maintained by many patients with chronic atrial fibrillation if they are adequately digitalized,¹⁰⁹ some of them do better when their rhythm is regular.³⁴ This is especially true during exercise. It has been demonstrated in these patients that cardiac output is increased and venous pressure decreased following conversion to sinus rhythm.¹⁴⁰ There is also the ever present danger that thrombi will form in the auricles of the fibrillating heart and that embolization will occur in these patients. For these reasons, we believe that an attempt to restore sinus rhythm should be made unless the heart is very large or there is a history of recent congestive failure. This opinion is not universally shared, however, and many physicians are satisfied merely to slow the ventricular rate with digitalis. It has been repeatedly emphasized in the literature that conversion to sinus rhythm by quinidine may result in embolization or death at the time of the change in rhythm,²¹⁹⁻²²² particularly

in patients with mitral stenosis and a large left atrium. This has not been our experience. Although it is often difficult, and occasionally impossible, to restore regular sinus rhythm in these patients, we have not experienced any serious accident during the "attempt."

When employed properly, quinidine is a safe and effective drug. The harmful effects following its use have, we believe, been overemphasized. Several premonitory signs and symptoms of overdosage in patients under quinidine treatment should be kept in mind. The most common of these may be grouped under the term "cinchonism." They include tinnitus, impaired hearing, headache, blurring of vision, giddiness, nausea, vomiting, abdominal cramps, and diarrhea. If the drug is given rapidly, by the intravenous route, respiratory depression and convulsions similar to those seen in animals may result.¹¹⁷ Rarely, a true idiosyncrasy may be present. In such cases acute respiratory distress and circulatory collapse appear even after small doses. Serious skin eruptions¹¹² occasionally are a manifestation of quinidine sensitivity and, rarely, thrombocytopenic purpura develops (*vide infra*).^{216, 201}

A dramatic instance of quinidine sensitivity was seen in P.K., a teacher of sixty-eight, who had had a coronary occlusion thirteen years ago and who recently experienced ventricular premature beats. On several occasions he developed acute laryngeal edema after receiving 3 grains (2 gm.) of quinidine twice a day. A single daily dose finally sufficed to prevent the premature beats and no further episode of edema occurred while the patient was on this dose. Another graphic demonstration of quinidine idiosyncrasy was that of S.G., a physician of fifty-six, with essential hypertension and coronary artery disease. On several occasions during the years he had received several grams of quinidine for ventricular premature beats. Six months ago he had taken 3 grams (2 gm.) of quinidine six times during a two-day period when he developed profuse

Arrhythmias

ecchymoses in the skin and mucous membranes and profuse hematuria. A blood count showed marked thrombocytopenia. Quinidine was discontinued and recovery occurred spontaneously within three weeks. These sensitivity reactions should be treated by immediate withdrawal of the drug. Blood transfusions should be given if required, ACTH and/or cortisone and antihistaminic preparations may be of great value.

Quinidine produces significant electrocardiographic changes, widening of the QT interval being the most common finding. This is caused by lengthening of the RT segment and, only occasionally, by widening of the QRS complex.²⁷ The latter indicates a toxic effect of quinidine. These changes were noted in a thirty-four-year-old man who was admitted to the hospital because of recurrent episodes of palpitation and was discovered to have a nodal tachycardia. He was put on quinidine, 6 grains (4 gm), every two hours. After 8 doses, an electrocardiogram showed a QT interval of 58 seconds, largely as a result of lengthening of the RT segment. When the quinidine was stopped, the QT interval gradually returned to within normal limits within twenty-four hours. The QT interval may be even more prolonged than in the case cited above if significant widening of the QT interval is found, or if some of the symptoms listed above become manifest, the drug should be discontinued. If the patient is desperately ill, however, because of an arrhythmia, it may be necessary to continue quinidine administration despite mild symptoms of toxicity. Even in these cases, if toxic symptoms increase in severity, the drug should be discontinued and other medication given.

Some authors have stated that quinidine should not be used in the presence of complete atrio-ventricular or bundle branch block,²⁸ but we have not hesitated to employ it in supraventricular arrhythmias with bundle branch block. We have not observed any serious reactions as a consequence of this therapy.

When quinidine is unobtainable, oral potassium (5 to 10 gm daily given in divided doses every four hours) may convert atrial fibrillation to sinus rhythm.²¹³ This therapy was employed during World War II with excellent results. "Potassium Triplex" provides a palatable liquid preparation.

In the treatment of acute paroxysmal fibrillation, occasional good results with Pronestyl (*vide infra*) have been reported.¹⁶⁷ This drug, however, is not successful in converting chronic fibrillation to regular sinus rhythm, although slowing of the ventricular rate may occur.

If heart failure is present during the paroxysm of atrial fibrillation, *digitalis* should be administered immediately. It increases the degree of A-V block and slows the ventricular rate, but usually does not affect the arrhythmia. The measures suggested below (Chapter 2) for the treatment of heart failure should also be instituted. Digitalization can be accomplished in many ways with various preparations. It is advisable to learn the potentialities of one preparation for oral use and of another for intravenous administration.

If the patient is acutely ill and immediate digitalization is indicated, Strophanthin K or Ouabain (G Strophanthin) should be given intravenously. These drugs have a very brief latent period and usually exert an effect in five to fifteen minutes, with a maximum effect in thirty minutes to two hours.^{31, 171} A safe initial dose of Strophanthin K is 0.3 mg and of Ouabain, 0.5 mg. Another 0.1 mg. may be given in half an hour and repeated every hour until the ventricular rate is slowed to 70 or less.³⁴² The total dose of either drug should not exceed 1.0 mg. in twenty-four hours. Strophanthin K is available in 1 cc. ampules containing 0.5 mg ("Strophosid") and Ouabain in both 1 cc ampules containing 0.1 mg and 2 cc ampules containing 0.5 mg. We have used both of these drugs intravenously with good results.

Lanatoside C (Cedilanid) may also be given intravenously. It has a slightly longer latent period than Strophanthin and causes a decrease in heart rate in from twenty minutes to two hours.¹² Its effect lasts longer than that of Strophanthin K or G, making this drug ideal if moderately quick and slightly prolonged action is required. The initial dose of 8 mg (4 cc) is approximately one half the digitalizing dose. This is followed by 4 mg (2 cc) every four to six hours, as required. Cedilanid is available in ampules of 2 cc containing 4 mg and in ampules of 4 cc containing 8 mg.

It is important to remember that these preparations are eliminated fairly rapidly, and that the full digitalization effect may be lost if a longer acting digitalis preparation is not administered as soon as practicable, i.e., within twenty-four hours. In this way, digitalization is maintained. It is usually not necessary to use intravenous preparations, except in patients with severe heart failure and rapid fibrillation.

For oral use, we have found Digoxin, a glycoside of *Digitalis lanata*, very satisfactory. It has the advantage of being a pure substance of definite and constant composition and, unlike digitalis leaf, it is almost completely absorbed from the gastro-intestinal tract.¹⁰ It is eliminated rapidly, and is not as apt to cause cumulative toxic effects, such as those produced by more slowly excreted glycosides, e.g., Digitoxin. The effect of a single dose of Digoxin usually persists only for several days,¹³ whereas the effect of a full dose of Digitoxin may continue for two to three weeks.¹¹ Digoxin is a safe and effective preparation for oral digitalization (Digoxin may also be used intravenously when a moderately rapid effect is required).

We have recently used Gitalin (Amorphous), a digitalis preparation which is also absorbed rapidly and completely and has a duration of action which is longer than Digoxin and shorter than digitalis leaf or Digitoxin. The major ad-

vantage of this compound appears to be the wide margin between the effective therapeutic and the toxic dose^{16,72} With most digitalis preparations the usual therapeutic dose is approximately two-thirds of the toxic one, with Gitahn the amount required for therapy is only approximately one-third of the toxic dose. We have employed this preparation in several patients who developed signs of "overdosage" while on Digoxin and have found that occasionally the use of Gitahn will make it possible to continue digitalization without toxicity. This is not true in all cases.

The amount of any digitalis preparation required for digitalization varies greatly from person to person, and the dosage must be individualized. The effective dose of Digoxin varies between 1.5 and 5.0 mg.⁷² An initial dose of 1.0 to 1.5 mg. is given, thereafter, 0.5 mg. is given every six hours, until the desired effect is obtained. The maintenance dose is .25 to .5 mg. (1 to 2 tablets) daily, most patients being well controlled on .25 mg. Frequently, giving .25 mg. and .50 mg. on alternate days proves very satisfactory. If Gitahn is used the patient may be rapidly digitalized by giving .25 mg. immediately and 1.0 mg. every six hours. Slow digitalization may be accomplished with this preparation by giving 1.0 mg. every four to six hours, or .5 mg. four times daily until the desired effect is obtained. The average effective dose of Gitahn is 6.0 and 7.5 mg., maintenance is usually achieved with .50 to .75 mg. daily.

Many physicians still use the whole digitalis leaf preparation for both digitalization and maintenance. This preparation is a good one, but the delayed onset of its action and the long period required for its dissipation are often disadvantageous. When the patient will not cooperate and take medication regularly, however, the use of the leaf is preferable, since the effect of Digoxin disappears in forty-eight hours and the patient may go into cardiac failure if he neglects to take it for two or three days. This danger is less when digitalis leaf is used. The whole leaf is much safer

vantage of this compound appears to be the wide margin between the effective therapeutic and the toxic dose.^{16 72} With most digitalis preparations the usual therapeutic dose is approximately two-thirds of the toxic one; with Gitalin the amount required for therapy is only approximately one-third of the toxic dose. We have employed this preparation in several patients who developed signs of "overdosage" while on Digoxin and have found that occasionally the use of Gitalin will make it possible to continue digitalization without toxicity. This is not true in all cases.

The amount of any digitalis preparation required for digitalization varies greatly from person to person, and the dosage must be individualized. The effective dose of Digoxin varies between 15 and 50 mg.⁷² An initial dose of 10 to 15 mg. is given, thereafter, 0.5 mg. is given every six hours, until the desired effect is obtained. The maintenance dose is 2.5 to 5 mg. (1 to 2 tablets) daily, most patients being well controlled on 2.5 mg. Frequently, giving 2.5 mg. and 50 mg. on alternate days proves very satisfactory. If Gitalin is used the patient may be rapidly digitalized by giving 2.5 mg. immediately and 1.0 mg. every six hours. Slow digitalization may be accomplished with this preparation by giving 1.0 mg. every four to six hours, or .5 mg. four times daily until the desired effect is obtained. The average effective dose of Gitalin is 6.0 and 7.5 mg., maintenance is usually achieved with 5.0 to 7.5 mg. daily.

Many physicians still use the whole digitalis leaf preparation for both digitalization and maintenance. This preparation is a good one, but the delayed onset of its action and the long period required for its dissipation are often disadvantageous. When the patient will not cooperate and take medication regularly, however, the use of the leaf is preferable, since the effect of Digoxin disappears in forty-eight hours and the patient may go into cardiac failure if he neglects to take it for two or three days. This danger is less when digitalis leaf is used. The whole leaf is much safer

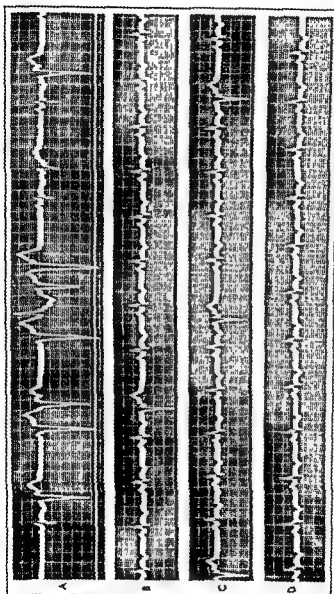


Fig. 1-5 W. Hypertensive cardiac disorder. A-Lead III 40 A, normal or premature beats per 100 beats. B-Lead II 15 minutes after 10 C in Potassium chloride. C-Lead II 30 minutes. D-Lead II 45 minutes. No premature beats. Courtesy of Dr. Charles J. Maclellan.

Table 3. Properties of Digital Preparations

	Dose and Action	Duration of Action	Duration of Effect	Maximum Dose	Comments
1. Neurohormonal A	Intermittent 5 to 15 minutes	24 to 48 hours	Average 16 to 18 mg	Not practical	Relief of spasticity (transient severe pruritus)
2. (Naloxone μ_2 Neurohormonal)	Weak effect 30 to 120 minutes				
3. Lamotrigine (L)	Intermittent 20 to 40 mg daily Weak effect (16 to 32) minutes	76 to 72 hours	Average 18 mg	4 mg 12 hours 4 to 6 hours	Intermittent action (severe pruritus)
4. Digoxin	1 to 2 hours Weak effect 4 to 6 hours	76 to 96 hours	Average 3.5 mg	2.5 to 3 mg daily	Local well tolerated usually short duration of symptoms
5. Clonidine (Neurohormonal)	1 to 2 hours Weak effect 4 to 6 hours	48 hours to 7 days	Average 6 to 7.5 mg	5 to 7.5 mg daily	Weak strong to intense (hypertension and lower dose)
6. Digitalis Leaf	1 to 2 hours Weak effect 6 hours	2 days to 2 weeks	1.2 to 2.9 mg	1 to 1.5 mg daily	Place above than safe and no response
7. Digoxin	1 to 2 hours Weak effect 4 to 6 hours	4 days to 2 weeks	1.2 to 2.0 mg	3.0 to 3.5 mg daily	High frequency of toxicity (severe pruritus)

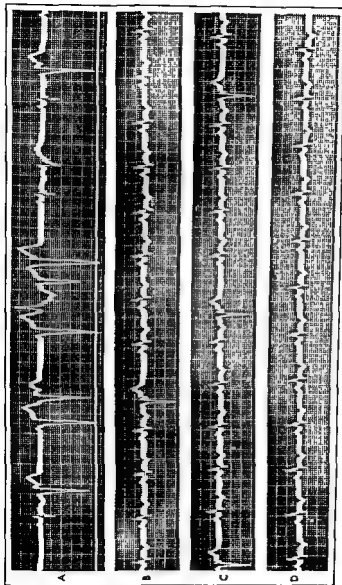


FIG. 1—SW Hypertensive cardiovascular disease. Digitals toxic. A—Lead III 40 Ventricular premature beats per 100 beats B—Lead II, 15 minutes after 10 Gm Potassium orally 6 premature beats per 100 beats C—Lead II, 30 minutes, 8 premature beats per 100 beats D—Lead II 45 minutes No premature beats (Courtesy of Dr Charles Enselberg)

arrhythmia except withdrawal of the drug. In some instances of supraventricular tachycardias,^{20, 21} ventricular premature beats, or ventricular tachycardias^{20*} induced by digitalis, specific treatment may become necessary. In these cases, potassium chloride or acetate, given orally, usually eliminates the arrhythmias within thirty minutes^{21, 22} (Fig. 1). Two to 10 grams of a 20 per cent solution may be given in wrap of citric acid or Karo syrup, or smaller quantities may be given in milk, orange juice, or ginger ale. Potassium Triplex (5 cc contains 5 gm each of Potassium Acetate, Potassium Citrate and Potassium Carbonate) is also an excellent way to administer potassium by mouth. Five to 10 cc are given in a glass of orange juice as needed. When potassium is given in this manner, nausea and vomiting usually do not occur. The effect produced will last only for from two to four hours, and the treatment may have to be repeated. Toxic effects of potassium do not occur if there is adequate urinary excretion. Rarely is it necessary to use potassium intravenously; if it is, extreme caution should be used, and only 5 to 15 gm should be given slowly. Electrocardiograms should be taken frequently to detect potassium toxicity. Marked peaking of the T-waves, widening of the QRS complexes, and disappearance of P waves are the most commonly observed changes, when potassium intoxication occurs.²²

Recent work clearly demonstrates that potassium depletion may sensitize the myocardium to the toxic effects of digitalis.^{19*} Conversely, maintaining an adequate cellular potassium may actually prevent toxic effects. For these reasons we usually give Potassium Triplex (1 teaspoonful, 5 cc, three or four times daily) to patients with myocardial disease in whom digitalis may have to be given in large doses. In this way arrhythmias may be prevented.

Atropine is often effective in abolishing the A-V block caused by digitalis.

It is often extremely difficult to distinguish between an arrhythmia and symptoms secondary to "overdigitalization" and those resulting from inadequately treated heart failure and "underdigitalization." For example, in patients who are receiving digitalis but remain in severe heart failure with a rapid ventricular rate and nausea and vomiting, it may be impossible to decide whether to withdraw the drug or administer larger doses. A procedure utilizing a rapidly acting digitalis preparation, acetyl-strophanthidin, has been introduced in an effort to determine whether or not a patient is adequately digitalized.¹⁸⁶ Since the effect of this preparation is noted within one to ten minutes after intravenous administration, the status of digitalization may be quickly determined. If a patient is adequately "digitalized" or "overdigitalized," toxic effects will usually appear after the injection of less than 6 mg of acetyl-strophanthidin. If they do not develop, overdigitalization is not usually the cause of the patient's symptoms or the arrhythmia. This preparation is extremely potent and several fatalities have resulted from its use. We prefer to use either intravenous Lanatoside C (Cedilanid), 4 mg every three hours, or Strophanthin K, .1 mg every thirty to forty-five minutes, to determine whether or not a patient is adequately digitalized. Although this method is slower, it is much safer and an answer to the problem is usually obtained within four to six hours.

In atrial fibrillation secondary to hyperthyroidism or active rheumatic fever, digitalis usually does not control the arrhythmia but occasionally has proven effective. For example, a forty-seven-year-old housewife was in severe congestive failure with atrial fibrillation and a ventricular rate of 170 to 180. She was given 8 mg Cedilanid, intravenously, and 4 mg, intramuscularly four hours later. She also received 2 cc, Mercurhydrin intramuscularly. The next morning atrial fibrillation was still present but her heart rate had slowed to 80 to 90. She had lost 8 pounds and

felt well. Radioactive iodine studies later definitely indicated hyperthyroidism to be the cause of her cardiac disability and she responded to treatment with the drug.

After the hyperthyroidism has been treated with radioactive iodine, operation or other drugs, the arrhythmia disappears spontaneously or responds to quinidine therapy.¹⁰ A case recently studied illustrates this point. A fifty-three-year-old female complained of constant palpitation. She had been found to have atrial fibrillation, and had received very large doses of digitalis and quinidine, without abolition of the arrhythmia or slowing of the ventricular rate. Although clinical signs of Graves' disease were absent, only 20 per cent of a test dose of radioactive iodine (I_{131}) was excreted and a diagnosis of hyperthyroidism was made. A therapeutic dose of 5 millicuries of I_{131} was administered. Two days later she was started on 30 drops of Lugol's solution daily. This medication was continued for four weeks and, while the fibrillation persisted, the ventricular rate fell to 84. The arrhythmia was still present six weeks after the administration of I_{131} and for this reason 6 grains of oral quinidine were given every three hours. Sinus rhythm appeared after 8 doses, and persisted even after quinidine was discontinued.

Radioactive iodine is usually picked up by the thyroid gland within twenty-four hours, but its effect on metabolic processes may not become evident for four to six weeks. It may be necessary, therefore, as in the case cited, to administer Lugol's solution (10 drops, t.i.d.) during this period. This medication may be started twenty-four hours after radioactive iodine has been given and should be continued for approximately three weeks. The effect of the Lugol's solution will last for another week, at which time the radioactive I_{131} effect will have begun to manifest itself.

If propyl thiouracil alone is used in hyperthyroid patients, an effect on the arrhythmia should be seen within two to four weeks.⁷ It should be emphasized that the prognosis in

those cases of atrial fibrillation due to hyperthyroidism is excellent, if the primary disease is controlled.

Cases of atrial fibrillation occurring during active rheumatic fever usually respond to digitalis or quinidine therapy only when the active process has been controlled. Failure to respond to digitalis suggests that the underlying disease is still active. The prognosis in these cases is not nearly as good as in the patients with hyperthyroidism, especially if a mitral valvular lesion is present.²⁵⁷

Inability to slow the ventricular rate with digitalis in patients with atrial fibrillation may also be noted in patients with metastatic malignancy involving the pericardium or atrial muscle. In these instances a mechanical factor produces stretching or irritation of the atrium and drug therapy is of no value.

ATRIAL FLUTTER

In *atrial flutter*, the ventricular rhythm is usually regular, with a rate of 125 to 180, but it may be irregular, because of varying degrees of A-V block, and thus may resemble atrial fibrillation. Atrial flutter may sometimes be distinguished from fibrillation by the observation of rapid, regular venous pulsations in the neck at a rate over 250, corresponding to the rate of the flutter waves in the electrocardiogram. Usually the atrial rate varies between 250 and 350, and 2:1 A-V block is present, but a marked degree or complete A-V block with very slow ventricular rate may occur. Exercise or position change may temporarily decrease the A-V block and increase the ventricular rate. Carotid sinus pressure does not abolish the flutter but often momentarily slows the ventricular rate.

When atrial flutter is complicated by a bundle branch block pattern, it may simulate ventricular tachycardia electrocardiographically (Fig. 2). Occasionally flutter can be diagnosed with certainty only in the electrocardiographic

leads taken to the right of the sternum,³¹ or from the right chest or back. In rare instances it is necessary to take esophageal leads to record the atrial waves.³²

Atrial flutter is usually paroxysmal, but it may become chronic and continue for months or years.³³ If the paroxysmal type does not remit spontaneously after a short period

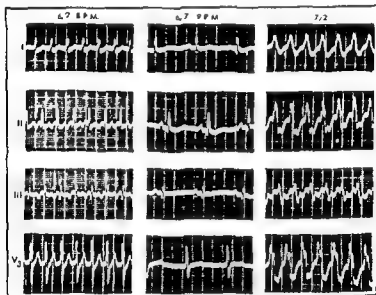


Fig. 2—5 W, m 71 Atrial flutter treated with intramuscular quinidine, 6 Gm., and oral Digoxin, 8 mg. 6/7 5 P.M. 2 1 atrial flutter 6/7 9 P.M. 1 hour after therapy, regular sinus rhythm 7/2 2 1 atrial flutter with bundle branch block, simulating ventricular tachycardia

of observation, an attempt should be made to stop the attack with digitalis and/or quinidine (Fig. 2). Digitalis is the drug of choice^{21 311 321} and is employed in all patients with heart failure. Digitalis slows the ventricular rate and increases the efficiency of the heart. It has been repeatedly

stressed, since the original observation in 1911,¹⁴⁰ that digitalis often converts atrial flutter to fibrillation and that sinus rhythm then follows withdrawal of the drug.¹⁴¹ Although recent observations³³ have cast some doubt upon this theory, we have seen this happen in many patients when digitalis is administered. In some instances digitalis apparently converts atrial flutter directly to sinus rhythm without an *observable* period of fibrillation. In other cases, flutter is converted to fibrillation by digitalis but normal sinus rhythm does not develop when the drug is withdrawn. In these cases quinidine should be administered in an attempt to convert fibrillation to sinus rhythm. Usually a small dose of quinidine is effective.

Digitalis and quinidine are employed as described under atrial fibrillation. In critically ill patients there should be no hesitation about using intravenous digitalis followed by intramuscular quinidine, if necessary. In some instances, atrial flutter persists in spite of all these measures, and the patient must be maintained on digitalis, as in chronic atrial fibrillation, in order to keep the ventricular rate between 65 and 75 beats per minute. Some patients may continue to have atrial flutter for years and then suddenly have the arrhythmia remit spontaneously.¹⁴²

If attacks of paroxysmal atrial flutter recur frequently, an attempt should be made to prevent them by the continuous use of quinidine. The dose of quinidine for maintenance is 3 to 6 grams (2 to 4 gm.), four times a day. In some cases, however, we have found it necessary to increase the dose and the frequency of administration, giving as much as 8 grams (6 gm.), four or five times a day. If this is not effective, digitalization should be carried out and a maintenance dose continued. In this way a certain degree of A-V block is maintained and, when an attack occurs, the ventricular rate may not markedly increase, despite a rapid atrial rate. This may serve to keep the patient symptom-free during an attack. This method is not

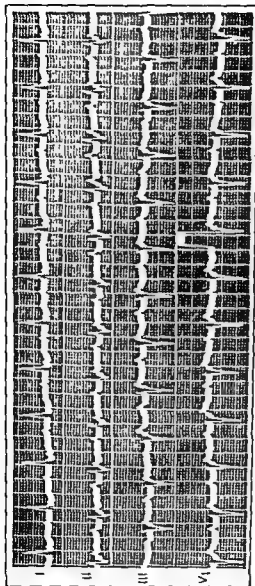


FIG. 3—1' E, III 52 Coronary artery disease. Paroxysmal atrial tachycardia with 2:1 A-V block. Atrial rate 100, ventricular rate 95.

successful in all cases and, despite adequate digitalization, a tachycardia may develop and symptoms appear

ATRIAL AND NODAL TACHYCARDIA

Atrial and nodal tachycardias cannot be distinguished clinically and are treated alike. These tachycardias are frequently functional and may be precipitated by tobacco, alcohol, infections, gastric distention, Graves' disease, allergic reactions and tense emotional states. Very frequently there is a combination of causes. The attack generally begins suddenly although in certain instances there may be a short premonitory period. The ventricular rate of 180 to 220 beats per minute usually is regular despite changes of position or exercise,⁸⁰ and is somewhat more rapid than in atrial flutter. There is usually a 1:1 ventricular response but occasionally a 3:1 or 2:1 response occurs with a resultant ventricular rate between 75 to 100.⁸¹ These cases are more common than was previously suspected (Fig. 3). Frequently a supraventricular tachycardia can be differentiated from atrial flutter by the absence of flutter waves in the cervical veins. Unlike cases of atrial flutter, in which only transient slowing of the ventricular rate may be noted following vagal stimulation, supraventricular tachycardia often is terminated by this procedure. These latter arrhythmias are particularly apt to be evanescent, and patients may have many attacks daily for years without experiencing difficulty. Patients subject to recurrent attacks have learned, through experience that certain maneuvers stimulating the vagus nerve abolish an attack, e.g., sudden movements of the head, holding the breath, coughing, vomiting, eating, lowering the head over the bed, or bending forward in a chair.

The attack often disappears spontaneously or following the use of sedatives but, if it persists, an attempt should be made to terminate it by exerting pressure on the carotid

sinus. This maneuver should be tried before other measures are employed. Pressure over the carotid sinus is applied with the patient in the prone position and with the head turned away from the side to be stimulated. The site to be pressed is recognized as a pulsating area at the angle of the jaw, at the level of the thyroid cartilage, here pressure is applied firmly towards the vertebral column. The right carotid sinus is usually more sensitive than the left.¹⁹³ Pressure should not be applied on both sides at the same time, especially in old people, and ten to twenty seconds should be the limit for pressure on either side. It should be stopped sooner if slowing of the heart rate occurs. Cases of syncope, convulsions and hemiplegia have been reported following carotid sinus pressure.⁴¹⁻⁴³ The prior administration of certain vagotomic drugs, such as Mecholyl³⁰¹ or Lanatoside C¹²⁹ augments the carotid sinus effect, while Benzedrine, epinephrine, neosynephrin, atropine, or large doses of quinidine may inhibit its effect.²⁴¹⁻²⁴³ If pressure on the carotid sinus fails at first it may be successful later, especially if it is applied in conjunction with one of the drugs which tend to increase its effect. It has been reported that carotid sinus pressure is effective in about 10 to 30 per cent of cases of atrial or nodal tachycardia,³⁰¹⁻³¹³ although others have had much more success with this method.¹²⁶ We have found it effective in about 25 per cent of our cases. Some observers believe that *carotid sinus pressure* or compression is not as effective as actual massage of the carotid sinus and they advocate this procedure.²⁴⁴ This method of therapy is potentially dangerous in elderly individuals.

Eyeball pressure, when employed properly, stimulates the oculocardiac reflex and produces inhibitory effects upon atrial muscle and atrio-ventricular conduction.¹²⁶⁷ Pressure for twenty or thirty seconds is applied over both eyeballs simultaneously, the fingers pressing on the closed eyes just below the supraorbital ridge, and not over the cornea.

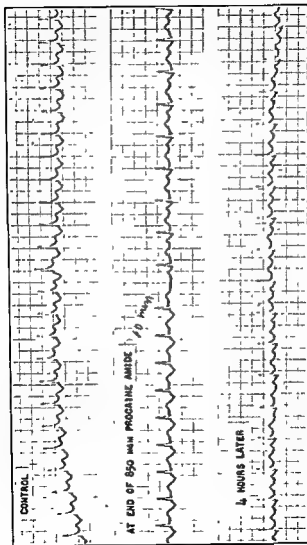


FIG. 4—M.B., m. 45. Paroxysmal nodal tachycardia of unknown etiology. Cessation 10 min. following 850 gm. procaine amide intravenously. Continuous lead II. (Kavden, Steele, Mark and Brodie, *Courtesy of Circulation*.)

Occasionally, a patient responds to eyeball pressure but not to carotid sinus stimulation. The danger of retinal detachment occurring in myopic individuals following this procedure has recently been stressed.²²

If the tachycardia persists despite these measures, quinidine, digitalis or procaine amide (Pronestyl)²³ should be used. Some authors prefer to employ digitalis routinely and it should, of course, be used in all cases of supraventricular tachycardia complicated by congestive heart failure, full digitalization is accomplished rapidly by mouth or vein as in atrial fibrillation or flutter. This drug appears to be most effective in the treatment of these tachycardias when given by the intravenous route. For example, excellent results have been obtained within ten to thirty minutes, with 1.2 mg of Lanatoside C (Cedilanid).¹⁴ Quinidine may be given orally or intramuscularly as previously outlined. It is effective in supraventricular arrhythmias in approximately 60 to 80 per cent of cases. Pronestyl, with an action similar to that of quinidine, is also effective in a large percentage of cases of atrial or nodal tachycardia. Figure 4 shows the rapid effect of .8 gm of the drug given intravenously to a man with nodal tachycardia. This patient of forty-five developed repeated episodes of this arrhythmia but no cardiac disease could be demonstrated.

Pronestyl is considered the drug of choice in atrial and nodal tachycardia by some observers²⁴ if carotid sinus pressure fails. Our own experience indicates that this drug offers few advantages over quinidine in supraventricular arrhythmias but occasionally is effective when the latter drug fails. This is illustrated by the case of a sixty-seven-year-old female with arteriosclerotic heart disease and repeated episodes of paroxysmal supraventricular (nodal) tachycardia who was admitted to the hospital with nodal tachycardia and a ventricular rate of 150. Sedatives, carotid sinus pressure and .8 gm of quinidine given over a period of 4 hours did not effect the arrhythmia. The next

morning intraventricular block was noted, although it was probably the result of the persistent rapid ventricular rate, it was considered best not to continue quinidine. 250 mg of Pronestyl were given intravenously and regular sinus rhythm was restored. The ventricular rate slowed to 85 to 90 and the intraventricular block disappeared promptly. The patient made an uneventful recovery.

Pronestyl may be given orally (250 to 500 mg every four hours), intramuscularly (as the hydrochloride) or intravenously (slowly with electrocardiographic and blood pressure control). Details of its administration are noted below.

In cases in which mechanical methods are unsuccessful, and in which more rapid action than is usually provided by quinidine is desired, *neosynephrine* may be employed. When given intravenously, in doses of 5 mg, this drug usually stops an attack of supraventricular tachycardia within twenty to thirty seconds.¹¹ It produces a rise in blood pressure, stimulation of the cardio-inhibitor fibers in the aortic arch and carotid body, and reflex cardiac slowing.¹² Most attacks revert when the systolic blood pressure has risen to 160 mm and the pressure usually returns to normal within ten minutes or less. There are few toxic effects of the drug but it should not be used in patients whose blood pressure is elevated during an attack or in cases with ventricular premature beats. We have found *neosynephrine* effective in several cases after all other methods had failed. Other pressor drugs such as *Vasoxyl* may also be used in treating supraventricular tachycardia.

If the above physical measures and drugs do not stop the tachycardia, vagal reflexes may be stimulated by inducing vomiting with syrup of ipecac, 4 to 8 cc by mouth.¹³ The dose may be repeated in forty-five minutes and larger doses may be given if necessary. This drug is highly successful in stopping an attack of tachycardia, but its unpleasant effects, i.e., severe nausea and vomiting, have

limited its use and we do not employ it unless absolutely necessary

Mecholyl (Acetyl beta methylcholine) is usually effective in treating supraventricular tachycardia, and restores normal rhythm within a few minutes,¹⁰³ but this drug, too, may produce serious and annoying side effects, and consequently we do not use it, unless other measures fail. It is injected subcutaneously, the initial dose being 20 milligrams. This may be repeated in twenty to thirty minutes. The effect of *Mecholyl* may be enhanced by massaging the site of injection and pressing the carotid sinus. The drug may cause severe nausea, vomiting, diarrhea, salivation, precordial pain or occasionally collapse. Shock and death¹ have been reported following its use. Therefore, it is absolutely essential that atropine sulphate, 1 to 2 mg (gr 1/60 or gr 1/30) be on hand when *Mecholyl* is used, since it relieves these symptoms immediately when given by vein. *Mecholyl* should not be given to allergic or asthmatic patients because of the danger of inducing bronchial spasm, or to hyperthyroid patients because atrial fibrillation may be precipitated.²⁴⁰ It is best to keep the patient in a horizontal position when this drug is used to avoid possible syncope. *Mecholyl* and carotid sinus pressure usually are ineffective in cases of atrial tachycardia with A-V block and in atrial flutter and fibrillation, and they may be ineffective if quinidine has been administered previously.³⁰¹

A less toxic parasympathomimetic drug which is effective in the treatment of supraventricular tachycardias is acetylcholine.¹ The effect of this drug is brief, and its side effects are less marked than those of *Mecholyl*. Although we have had little experience with this preparation, other observers¹ have found it to be satisfactory when given in a single intravenous dose up to 100 mg.

Neostigmine, another parasympathomimetic drug, is also effective in abolishing supraventricular tachycardias and is given in doses of 1 mg intramuscularly.³²¹ When any of

Table 4. Differential Diagnosis and Treatment of Paroxysmal Tachycardia

Type of Arrhythmia	Other Diseases Patient	ECG Heart Rate—Pulse Rate Symptoms	Treatment*
(A) Acute Paroxysmal Atrial Fibrillation	(1) Rheumatic by persistence of atrial fibrillation (2) Coronary disease (3) Gravid disease (4) Infectious postoperative etc	Heart rate 120-180, pulse definite may be present Pulse rate 70-120 or more, completely irregular Occasional escape	(1) Sedation (2) Quinidine (3) Digoxin (4) Potassium
(B) Atrial Flutter	(1) Most common in rheumatic and arteriosclerotic heart disease (2) May occur in normals	Pulse rate 125-150 depending on degree of heart block, rate varies as degree of block changes Flutter waves in lead at rate of 250-300	(1) Digoxin until fibrillation then stop, or continue to keep slow rate (2) Quinidine (3) Potassium
(C) Supraventricular Tachycardia (a) Atrial Tachycardia (b) Nodal Tachycardia	(1) Patient usually normal (2) In organic heart disease (3) Postoperative (4) Gravid disease	Palpitations, distress and precordial pain may occur Rate 150-250	(1) Sedation (2) Vagal stimulation by carotid sinus pressure, etc. (3) Drugs: Acetyl salicylate, digitalis or quinidine if no effects, Atropine, Acetylcholine etc
(D) Ventricular Tachycardia	(1) Usually patients with or after coronary atherosclerosis (2) Digitalis intoxication (3) Heart failure	Occasional escape Rate 150-250 Blood pressure low to shock level Variations of intensity of first heart sound Early prominent neck pulsations	(1) Potassium, 1 v, 1 m or by mouth (2) Quinidine, 1 m, or by mouth
(E) Ventricular Fibrillation	(1) Usually patients with arteriosclerotic heart disease	Shock - Adams occurs common Pulse undetectable Heart rate not obtainable Blood pressure—shock level	(1) 1 v Quinidine—must be used in complete heart block (2) 1 v Potassium—must be used in complete heart block (3) 1 v Atropine (4) 1 v Atropine

* See text for complete details

these drugs is used atropine should be available for immediate use.

It should be remembered that supraventricular tachycardias so often remit spontaneously that the accurate appraisal of the efficacy of any drug in their treatment is difficult. If there are numerous recurrences of the tachycardia, the frequency of the attacks may be diminished by the continued use of quinidine, Pronestyl or digitalis.

A review of the common clinical features of the supraventricular arrhythmias is presented in Table 4.

VENTRICULAR TACHYCARDIA

Ventricular tachycardia is infrequent and is usually associated with severe coronary artery disease and coronary occlusion. It may be seen in cases of rheumatic heart disease, during cardiac catheterization or operation, and occasionally in normal persons.³ Its occurrence in patients with the Wolff-Parkinson-White syndrome is not uncommon. Occasionally it is possible to differentiate ventricular tachycardia clinically from atrial tachycardia by a slight irregularity in rhythm and a variation in the intensity of the first heart sound. The cardiac rate should be counted over a period of two or three consecutive minutes. If the rhythm is basically regular at a rate of 150 to 250 but there is a rate variation (3 to 10 beats) from minute to minute the tachycardia is probably ventricular rather than atrial or nodal. Prominent jugular vein pulsations, representing a summation of the atrial systolic wave (A wave) and the ventricular systolic wave (C wave) may be observed at a rate slower than that at the apex, although this is not a common finding.

Digitalis is often a factor in the production of ventricular tachycardia.³³³ Some observers believe that all cases of the bidirectional type of this arrhythmia are secondary to digitalis overdosage.¹⁹⁰ We have stressed the fact that digitalis

should be discontinued in all patients who develop ventricular premature beats, especially if they are multifocal. If this precaution is taken, many cases of ventricular tachycardia can be prevented.

Ventricular tachycardia usually responds to quinidine or Pronestyl. Quinidine may be given orally or intramuscularly occasionally, an intravenous dose, (1 gm, 15 grains) has been required to stop an attack.²⁴ We believe, however, that quinidine should be used intravenously only if absolutely necessary because of its toxicity when given by this route, and because intravenous Pronestyl is safer if an immediate effect is required.

Pronestyl, a procaine derivative, is very effective in the treatment of ventricular tachycardia.^{20a} It is a direct myocardial depressant with an action similar to that of quinidine. Pronestyl is supplied in 250 mg capsules for oral use, and in ampules containing 100 mg per cc for intravenous administration. We have used it with excellent results in ventricular tachycardia and premature beats, including cases in which quinidine has failed. It is given orally in doses of 500 mg every three to four hours, or intravenously in doses of 250 mg to 1 gm at a rate of 100 mg per minute every thirty to sixty minutes until an effect is obtained. An electrocardiogram should be taken while Pronestyl is being given intravenously as it is often effective immediately. If a change in rhythm or widening of the QRS complex occurs, the drug should be stopped. Occasionally oral doses up to 6 or 10 gm are required. We have found that both routes of administration are satisfactory for the treatment of the acute episodes but intravenous administration produces a much more rapid effect. Ventricular tachycardia may be converted to regular sinus rhythm in from thirty seconds to two minutes following as small a dose as 250 mg of Pronestyl. A case recently observed is of interest to illustrate the effect of this drug.

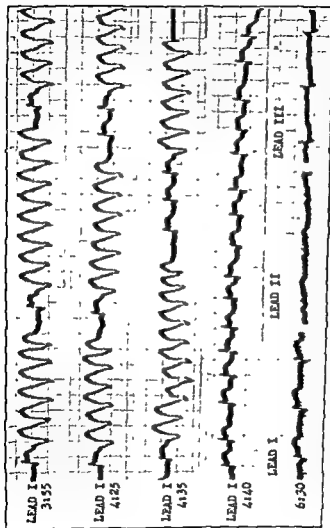


FIG 5—M Y, m 66 Acute coronary occlusion. Precordial pain, heart failure and occasional palpitation 16 days. Strophanthin E 4 mg just prior to admission. From st 10 gm orally 3:45 p.m. Digoxin 1.0 mg orally at 6 p.m.

A sixty-six-year-old white male experienced an attack of squeezing, severe chest pain radiating to the left shoulder and arm ten days prior to hospital admission. Four days prior to admission he developed dyspnea, on the day of admission he became dizzy and weak, noted palpitations, and fainted. On admission, he was in acute pulmonary edema and was given 4 mg Strophanthum K intravenously, morphine, and 2 cc Mercuhydram intramuscularly. The electrocardiogram showed runs of ventricular tachycardia and acute anterior wall infarction (Fig 5). He was given 1 gm. of Pronestyl by mouth and normal sinus rhythm developed within an hour (Fig 5). The Pronestyl was continued in doses of 1 gm every four hours for two days, and then in doses of 5 gm every four hours for eight days. He was also digitalized with Digoxin and made an uneventful recovery.

Pronestyl generally does not produce serious reactions, but when administered intravenously it may cause a marked fall in blood pressure or even cardiac standstill. The injection should be given slowly and blood pressure readings should be made during the injection. If a fall in blood pressure does occur, it may be counteracted by Neosynephrin (1 to 5 mg) intramuscularly (25 mg Neosynephrin may be given prophylactically with the Pronestyl). When given orally in large doses Pronestyl may produce nausea and other gastric symptoms making it necessary to stop therapy or reduce the dose. Fever, chills, malaise and skin rashes have also been noted following its use.¹⁴ We have observed one patient who developed agranulocytosis while receiving Pronestyl and this has recently been pointed out by others. Frequent blood counts should be taken and evidence of purpura or bleeding watched for.

Pronestyl is occasionally useful in the prevention and treatment of ventricular arrhythmias associated with operative procedures¹⁴ although some observers doubt its effectiveness. If used preoperatively, 1 to 2 gms may be given

by mouth forty-five to sixty minutes prior to surgery, and 1 gm given intravenously as the operation is begun. During anesthesia and operation, intravenous procaine may be used instead in a dose of 5 cc of a 1 to 2 per cent solution, to prevent ventricular arrhythmias.⁴⁹

Pronestyl has now been used prophylactically by us in a small series of patients who had previously experienced repeated bouts of ventricular tachycardia. Although it terminates the acute attack effectively, it does not appear to be completely effective in preventing recurrence of attacks when used orally, 500 mg, 3 to 4 times daily. It may be that this dose is inadequate, and patients with repeated attacks may require much larger doses. At the present time the best procedure appears to be to give Pronestyl for the acute episode and quinidine prophylactically.

Many other drugs that have been used in the treatment of ventricular tachycardias deserve mention at this time. One of these agents, intravenous magnesium sulfate, is not widely used, but has proven effective in occasional cases.⁵⁰ When given rapidly, in 2 to 4 gm doses, i.e., 10 to 20 cc of a 20 per cent solution, this drug may terminate an attack after other measures have failed. Transient, unpleasant side effects such as nausea, flushing, weakness, and dizziness may appear. It should be reserved for use after other therapy has proven ineffective. With the new agents available, little opportunity for the use of magnesium sulfate presents itself. Atabrine, in doses of .4 gram intramuscularly, may occasionally be effective.¹¹³ Intravenous morphine sulfate, 10 to 20 mg (1/6 to 1/3 gr), repeated every hour if necessary, has also been used successfully by several observers.¹²²⁻¹⁴⁰

The use of potassium salts (citrate or acetate in syrup or water, or chloride as enteric coated tablets) orally, either alone in doses of 1 to 5 gms two to four times daily, or in conjunction with quinidine, has been advocated for the

treatment of refractory cases, or to diminish the frequency of recurrences of ventricular tachycardia.¹⁰⁷ We have found potassium to be particularly useful in the treatment of ventricular tachycardia secondary to digitalis overdosage and in patients who are sensitive to quinidine. Dibenamine hydrochloride, Dibenzylne and Regitine, specific adrenergic blocking agents, have been shown to be effective in blocking arrhythmias which occur during cyclopropane anesthesia,¹⁶¹⁻¹⁶⁵ but we have not used them in the treatment of tachycardias.

VENTRICULAR FIBRILLATION

Ventricular fibrillation is rarely diagnosed clinically, and, although it probably accounts for some cases of sudden death, it is difficult to state its exact incidence. Many Stokes-Adams attacks are the result of ventricular fibrillation, in fact, this arrhythmia is frequently noted in patients with varying degrees of atrioventricular block. Most episodes of ventricular fibrillation occur in association with myocardial infarction, anesthesia, electric shock or excessive amounts of digitalis, quinidine, Pronestyl or other myocardial depressants or stimulants. The patient becomes pulseless, unconscious and rapidly goes into shock. Convulsions may occur if this arrhythmia persists for more than thirty seconds. The electrocardiogram shows bizarre ventricular complexes and a completely irregular rhythm. *Clinically*, these attacks resemble a Stokes-Adams seizure secondary to complete heart block and cardiac standstill and it is important to distinguish between these conditions electrocardiographically. The use of sympathomimetic drugs such as epinephrine or ephedrine, which are so valuable in the treatment of cardiac asystole, is contraindicated in ventricular fibrillation. If possible, the physician should obtain an electrocardiogram during the attack to establish its exact mechanism. Once an episode is diagnosed correctly

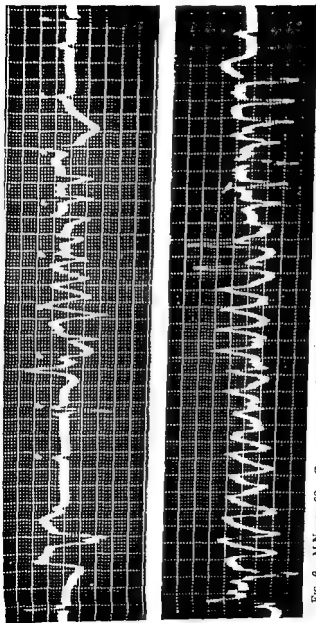


FIG 6—M N., m 60 Coronary artery disease Complete A-V block with periods of ventricular fibrillation and Stokes-Adams seizures Lead II continuous

as ventricular fibrillation, it can be assumed that subsequent attacks will be caused by a similar mechanism and not by cardiac standstill, and they may be treated accordingly. Unfortunately this is not always the case as bouts of both ventricular fibrillation and cardiac asystole may occur in the same patient (Fig 6)

The treatment of ventricular fibrillation during operative procedures is discussed in detail in Chapter 9. Treatment of this arrhythmia when it occurs secondary to other factors is by no means standardized. Oxygen should be administered immediately and intravenous procaine given. If this is not available either procaine amide (Pronestyl) (100 mg/minute) or quinidine (4 to 6 gm) is given intravenously, despite the fact that this route of administration is not without danger. If either drug is given carefully with electrocardiographic control, an occasional good result may be obtained. *Neither drug should be used in patients with previous, known complete heart block.* Pronestyl is probably the safer drug. The cautious administration of intravenous magnesium sulfate (10 cc. of a 20 per cent solution) or potassium chloride is also occasionally satisfactory. Intravenous atropine, 2 mg (gr 1/30), has been used successfully to abort the acute attack, and smaller doses, 5 mg (gr 1/100), intramuscularly have been given daily to prevent recurrent bouts in patients subject to paroxysmal ventricular fibrillation²⁵⁷. In many cases the attack will cease or the patient expire before therapy can be instituted.

HEART BLOCK

Complete heart block, with sudden failure of the idioventricular pacemaker and asystole, is the most common cause of Stokes-Adams syndrome although a similar clinical picture may be seen in cases of ventricular tachycardia or other arrhythmias²⁵¹. Heart block is usually seen in patients with organic heart disease secondary to coronary arterio-

sclerosis, but a small percentage of cases results from digitalis overdosage. Many patients experience only momentary weakness or unconsciousness and require no therapy, but others have severe symptoms and require immediate treatment.

If asystole is established as the cause of an episode of unconsciousness, vigorous thumping on the precordium may immediately restore cardiac action. This simple procedure has proved effective in many instances.^{25, 26} If this does not produce a satisfactory result, one-half of one cc of a 1:1000 solution of epinephrine may be given intravenously, or directly into the heart, in extreme cases in shock. If the seizures recur, the Adrenalin should be repeated subcutaneously every hour or two, or may be given in oil (1 cc = 2 mg) intramuscularly, every twelve to twenty-four hours. We have observed a fifty-six-year-old female who required 5 cc of Adrenalin every fifteen to thirty minutes for hours, in order to remain free of attacks. Intracardiac Adrenalin was necessary on one occasion after intramuscular therapy had failed, and during a period when the patient experienced recurrent Stokes-Adams seizures, a slow, continuous intravenous injection of 1:100,000 Adrenalin at a rate of 20 cc (30 drops) per minute was maintained for several hours in order to stop the syncopal episodes. In using epinephrine for the treatment of asystole, care should be taken not to administer it too rapidly since ventricular tachycardia or fibrillation may result following relief of the asystolic phase. In patients who experience frequent episodes of Stokes-Adams seizures, ephedrine gr 3/8 to 3/4 (25 to 45 mg) or Paredrine gr 1/3 to 10 (20 to 60 mg) may be given orally three to five times daily in an effort to decrease the number of recurrences.²⁷ Sublingual Isuprel (10 to 15 mg every four to six hours) has been of value in many cases. In all cases where digitalis may be a contributing factor this drug should, of course, be withdrawn. Some observers have achieved results in the treatment

of patients with complete heart block and ventricular standstill by the use of an externally applied cardiac *pacemaker*²⁴⁶ The pacemaker is not effective in treating syncopal attacks due to paroxysmal ventricular tachycardia or fibrillation

OTHER ARRHYTHMIAS

Sinus bradycardia, sinus tachycardia and premature beats may occasionally cause severe difficulty These will be discussed briefly

Sinus tachycardia is usually associated with febrile or toxic conditions, congestive heart failure, or emotional or metabolic disturbances There is no specific therapy other than that directed at the primary condition This should be carefully searched for and treated Occasionally dramatic results may be obtained For example a seventy-year-old woman who had experienced almost nightly episodes of persistent sinus tachycardia (rate 130 to 150) had been treated for one year with intravenous sedatives and Demerol during each attack Severe angina occurred and the patient was almost a total invalid It was demonstrated that each bout of tachycardia was closely related to painful bowel movements associated with large hemorrhoids and that the resting pulse rate of 100 to 110 in between attacks was most probably the result of hyperthyroidism (radioactive I_{131} uptake of 37 per cent) Following therapy with propylthiouracil and removal of the hemorrhoids, all attacks ceased, angina completely disappeared, and the patient did remarkably well

Sinus bradycardia, like sinus tachycardia, may be an incidental finding, especially in athletes and pregnant women, and usually requires no treatment It is caused by an increase in vagal tone, or by a diminution in sympathetic tone, or both²⁴⁷ If the rate falls below 40, dizziness and syncope may result, as in a person with carotid sinus syn-

drome. Actual cardiac standstill may occur.²¹³ We recently observed a basketball coach of thirty-two who usually had a pulse rate of 48 beats per minute. He fainted on several occasions during tense moments in a game. During the attacks of syncope his pulse rate fell to 30, but he usually recovered after thirty to forty seconds. Usually these cases respond to ephedrine in 25 to 45 mg ($\frac{1}{2}$ or $\frac{3}{4}$ gr.) doses, or to atropine sulphate (.4 to .6 mg) gr 1/150 to gr 1/100 by mouth. It may be necessary to give atropine (.6 mg to 2 mg) gr 1/100 to 1/30 subcutaneously or intravenously, if an immediate effect is desired.

Premature beats may cause palpitations, precordial discomfort and anxiety, particularly if they occur frequently. These symptoms can usually be relieved by reassurance, rest and sedation (Phenobarbital, .06 to .03 gm four times daily), but occasionally they are sufficiently severe and persistent to require active treatment. A definite relationship has been found between anxiety states and episodes of premature beats, and it has been shown that they, as well as the distressing symptoms which occur during attacks, may, in some cases, be eliminated by adequate psychiatric therapy.²¹⁴ Use of alcoholic beverages, excessive smoking or, in some individuals, even minimal smoking, may produce annoying episodes of "palpitations" as a result of premature ventricular contractions. All smoking should be interdicted in these patients. If drugs are necessary to stop premature beats, the physician should determine the site of origin of the ectopic beats before beginning therapy. Quinidine is usually successful in reducing the frequency of both ventricular and atrial premature beats. Pronestyl (procaine amide) is more effective in stopping or preventing ventricular premature beats than atrial premature beats. For example: a forty-two-year-old salesman began to experience frequent choking sensations associated with a momentary cessation of the heart beat and, on several occasions, he became faint and almost collapsed. Examina-

tion showed the heart to be normal but the electrocardiogram revealed ventricular premature beats. He received 250 mg of oral Pronestyl 4 times a day with prompt disappearance of the arrhythmia and has remained symptom-free since that time.

Although this patient was controlled by 250 mg of Pronestyl four times daily, usually at least 500 mg is required orally every four to six hours to attain a similar effect.

Premature beats assume great clinical importance when they precede the onset of paroxysmal tachycardia, especially during operative procedures or following a coronary occlusion.

Chapter

2

ACUTE PULMONARY EDEMA AND CONGESTIVE HEART FAILURE

MANY previously accepted concepts of cardiovascular and renal physiology and of sodium metabolism have been altered and new concepts of the pathologic physiology of heart failure and acute pulmonary edema have been introduced during the last ten years

Pathogenesis of Cardiac Failure.—In the past, the symptoms and signs of congestive failure were attributed to "backward" failure, *i.e.*, myocardial insufficiency (cardiac failure), with increased venous pressure and venous congestion, increased blood volume secondary to anoxia of the bone marrow, and resulting edema ¹⁰⁰ Recent investigations, however, have shown that this mechanism does not explain many of the findings in congestive failure. These observations support the theory of so-called "forward" failure, *i.e.*, cardiac insufficiency, diminished cardiac output and renal blood flow and decreased sodium excretion, with resulting increase in blood volume and edema ¹¹¹ Most of the symptoms and signs found in congestive failure can be accounted for by this sequence of events although they do not explain some of the findings in acute heart failure, *i.e.*, acute pulmonary edema (*vide infra*) When cardiac output falls, there may be a liberation of excessive amounts of antidiuretic hormone from the posterior pituitary gland ²⁷ and of desoxycorticosterone from the adrenal cortex, ²¹³ these

also play a part in the retention of sodium and water and contribute to edema formation. Although the pathogenesis of congestive heart failure has not yet been completely clarified, it is probable that elements of both the "forward" and "backward" hypotheses play a part in its development.

Excellent reviews dealing with the mechanisms of cardiac failure have been written.^{64 248 267 302} We wish, however, to emphasize certain facts presented in these papers concerning the patho-physiology of the various types of heart failure. A knowledge of these basic facts is essential for the intelligent handling of both the acute and chronic forms of this condition.

Heart failure occurs when the myocardium is no longer able to pump sufficient blood to all the tissues to satisfy their metabolic requirements. At first, the diseased myocardium is able to respond to stresses by increasing the length of its muscle fibers and contracting more forcefully (Starling's law of the heart).³¹⁰ Eventually, however, the fibers are distended beyond physiological limits and are no longer able to maintain an adequate output for tissue needs.

In congestive failure, the cardiac output may be low or normal as in hypertensive, arteriosclerotic or rheumatic heart disease, or it may be normal or high, as in thyrotoxicosis,³⁰³ anemia,³⁰⁵ beri beri,⁶¹ Paget's disease,⁷⁸ arteriovenous fistulae⁵³ or chronic pulmonary disease.²⁶⁸ In these latter conditions, although the output is not decreased, it still is unable to meet the tissue needs.³⁰⁴ A primary object in therapy, therefore, is to increase the cardiac output. In cases of failure with low cardiac output this can often be done by decreasing the blood volume by means of salt restriction or phlebotomy. The resultant diminished venous return and lowered venous pressure enable the overdistended, inefficient cardiac muscle fibers to shorten to within physiological limits, to contract with greater force, and to increase stroke volume and output.

In cases of failure with high output, correction of the primary condition usually brings relief.

Digitalis increases cardiac output in both types of heart failure although it is probably more effective in cases with low output.¹⁰² The drug acts directly on the heart muscle, increasing the force of its contraction,^{76 118} although the exact mechanism of the cellular change which improves myocardial contractility has not been determined. A primary effect on the venous system has been suggested⁷⁴ but this observation has not been confirmed.

Sodium retention is probably the most important factor in the production of the *symptoms* of congestive failure. The retention of sodium and the consequent increase in total blood volume has been attributed to diminished cardiac output, diminished renal blood flow²²¹ and decreased renal excretion of sodium and water.²²⁴ A patient with heart disease may remain well compensated while his sodium intake is restricted but may quickly go into cardiac failure if given salt, the resultant increase in blood volume throws an additional load upon the already overburdened heart.²¹⁶ Adequate salt restriction and the use of mercurial diuretics to aid sodium excretion are, therefore, of prime importance in the management of congestive failure.

A marked increase in body activity increases the work of the heart and may cause a patient who was well compensated during rest or mild activity to go into severe cardiac failure. It is important, therefore, to regulate a patient's activity carefully in order to avoid sudden episodes of cardiac failure.⁷ The importance of adequate rest, either in a chair or in bed, especially during the initial period of treatment of heart failure, cannot be emphasized too strongly.

If the above principles are borne in mind in the course of treatment of congestive failure, many lives will be saved and careless mistakes avoided, for example,⁷ phlebotomy is of great value in acute left heart failure secondary to hypertensive heart disease,¹⁴⁰ i.e., cardiac failure with a low

output, but it may cause great harm if performed in other forms of heart failure, *e.g.*, heart failure associated with chronic anemia or arterio-venous fistulae. In these latter conditions, a high output is necessary to maintain adequate tissue oxygenation, and measures that lower the blood volume, such as phlebotomy, should be avoided since they further decrease the amount of oxygen available to the tissues.

Decreased renal blood flow, sodium retention and increased blood volume account almost entirely for the symptoms and signs of chronic congestive heart failure, *i.e.*, dyspnea, orthopnea, pulmonary congestion, distention of the neck veins, hepatomegaly, ascites, and peripheral edema. Additional factors must be sought, however, to explain the sudden occurrence of acute left heart failure, *i.e.*, cardiac asthma and acute pulmonary edema.

Paroxysmal Nocturnal Dyspnea.—Many patients with mild left ventricular failure experience recurrent episodes of paroxysmal dyspnea several hours after they retire. They awaken suddenly and are acutely dyspneic, a few rales and pronounced wheezes appear over both chests, without other evidence of failure. The blood pressure is high and the pulse is strong. Breath sounds may be markedly diminished or absent over portions of the lung fields because of intense reflex bronchospasm. Paroxysmal nocturnal dyspnea, commonly called cardiac asthma, is probably the result of a redistribution of blood with an increase in blood volume when the patient assumes the recumbent position. During the day blood is pooled in the splanchnic and peripheral areas but when the patient lies down, blood is redistributed to the thorax⁷⁹ and lung congestion results. As a result of this congestion, nerve reflexes from the lungs are initiated, and bronchospasm, dyspnea, and the other symptoms and signs of cardiac asthma result. The exact mechanism of this condition has not been completely clarified.

Complete relief is often obtained by having the patient sit up, stand, or walk about, if possible. Mild cases may be controlled by aminophyllin suppositories (.5 gm., 7½ grains). This drug is safe when thus administered and is completely absorbed, and large doses may be given without producing significant discomfort. The peripheral resistance is high in many of these cases because of sympathetic nerve discharge, and it may be necessary to give the aminophyllin intravenously (5 gm. slowly).¹⁵⁶ In the more severe cases which do not respond to this drug, morphine sulfate should be given intramuscularly, 15 mg ($\frac{1}{4}$ gr), or intravenously, 8 to 10 mg ($\frac{1}{8}$ to $\frac{1}{6}$ gr). The intramuscular route is usually satisfactory. Morphine usually very effectively suppresses the pulmonary reflexes that serve to perpetuate symptoms. If the attack persists, further treatment as outlined below for pulmonary edema should be carried out.

Recurrences of cardiac asthma can usually be prevented by the usual measures employed in congestive failure, *i.e.*, a low sodium diet, digitalization and mercurial diuretics, and by having the patient sleep on several pillows or in a chair, in the severe cases. In some of the milder cases digitalization may not be required.

Acute Pulmonary Edema.—The onset of acute pulmonary edema secondary to left ventricular failure may be extremely sudden with signs of circulatory collapse, severe dyspnea and cyanosis. It may occur following exercise, a large meal, paroxysmal tachycardia, a coronary occlusion, trauma to the central nervous system, or after severe emotional upsets.¹⁴⁷ A discharge of impulses from the sympathetic nervous system has been suggested as a possible explanation in the latter conditions.¹⁰⁷ Occasionally pulmonary edema may complicate bulbar poliomyelitis. For this reason intravenous hydration should be carefully supervised in these cases.³²⁷

A good example of pulmonary edema induced by neurogenic factors is illustrated by a sixty-five-year-old woman who had experienced occasional attacks of pulmonary

edema three years before but had been quite well since then. She recently witnessed her husband fall as a result of a cerebral hemorrhage and severely injure his scalp. He quickly developed a left hemiplegia. His wife did not appear anxious, but an hour later she suddenly began to wheeze and numerous rales appeared in both lungs. She was given Demerol, 50 mg subcutaneously, and gradually improved.

During the acute attack the heart sounds may be obscured by numerous bubbling rales and wheezes. In many cases the right heart is strained by the failing left ventricle and the pulmonic second sound is accentuated. When the patient is seen early, the blood pressure and pulse volume are often found to have been maintained. A short time after the onset of symptoms, however, there is a marked fall in blood pressure and the pulse becomes rapid and thready, i.e., shock occurs as a result of left ventricular failure. Morphine sulfate should be given intramuscularly at once, or intravenously in cases with peripheral collapse in which little or no absorption would otherwise occur. The doses employed are similar to those given in "cardiac asthma." This drug usually produces a dramatic improvement, but if no effect is noted, it should be repeated in fifteen to twenty minutes. Dilaudid, 2 to 3 mg ($1/32$ to $1/20$ grain), Pantopon, 20 to 30 mg ($1/3$ to $1/2$ grain), or Demerol (100 to 150 mg.) may be used instead of morphine, especially if the latter induces vomiting. Although some physicians use atropine, 0.4 to 0.6 mg ($1/150$ to $1/100$ gr.), routinely in the treatment of acute pulmonary edema, we do not believe that it is of value, in cases in which tachycardia is present it may actually be harmful.

Despite the fact that the arterial blood is fully oxygenated in many cases, oxygen is of value in pulmonary edema.¹⁴ Often, when given by the meter mask under pressure (3 to 6 cm.), it produces dramatic results. For example, A thirty-year-old negro, para iii, with rheumatic heart dis-

case, experienced mild toxemia of pregnancy. She was delivered of a full-term normal infant after a short labor but bled profusely and rapidly went into shock. She received 500 cc. of plasma and 2500 cc. of blood within the next three hours after having lost an estimated 2000 cc. of blood. She recovered from shock but developed marked pulmonary edema one hour later. Despite repeated doses of morphine, oxygen by tent, 8 mg. (4 cc.) Lanatoside C intravenously, and continuous bloodless phlebotomy with tourniquets, (*cule infra*), she did not improve. After one hour of vigorous therapy, oxygen under pressure (4 cm.) was administered by the anesthesiologist and within ten to fifteen minutes marked improvement occurred. The rales gradually disappeared and the patient recovered.

To prevent recurrences oxygen may be given in this way for a period of fifteen minutes every few hours for ten to twelve hours after the episode of pulmonary edema has been controlled. An early recurrence is occasionally prevented by this therapy. Oxygen by nasal catheter usually is not satisfactory, but an oxygen tent often is of value in warm weather. It should be remembered that the percentage of oxygen inspired by a patient when a nasal catheter or tent is used is never so great as that inspired through a mask (35 to 65 per cent as compared with 50 to 100 per cent). There is some evidence that 100 per cent oxygen may be irritating to the lung parenchyma, particularly if taken for hours or days, for this reason the use of a lower concentration is advisable.¹⁸²

Intravenous aminophyllin, 0.5 gm. (7½ gr.), is of particular value in the treatment of acute pulmonary edema when Cheyne-Stokes respiration is present. When given by vein it should be administered very slowly, the total dosage being injected in forty-five to sixty seconds.

If the pulmonary edema persists in spite of the above measures 3 mg. of *Strophanthin K* or .5 mg. *Ouabain* (*Strophanthin G*) should be given intravenously. *Cedilanid*

(Lanatoside C) or Digoxin may also be used intravenously (the Digoxin preparation presently available may be used without diluting) Two cc of a mercurial diuretic should also be administered. Thereafter, the Strophanthin is repeated if necessary or another digitalis preparation is used as outlined previously (Chapter I) When digitalis is given in elderly persons, the dosage employed should be less than in younger patients since sensitivity to the drug apparently is greatly increased in the aged. Mercurial diuretics may be more toxic when used intravenously¹⁶⁶ but should be given by this route in acute emergencies, e.g., when a patient is in shock and absorption is poor by other routes, the intramuscular or subcutaneous route should be employed thereafter.

Bloodless phlebotomy by means of tourniquets,¹⁷⁸ if employed properly, may be effective in patients with pulmonary edema secondary to low output cardiac failure.¹⁹⁷ Tourniquets are placed on three extremities and sufficient pressure is exerted to occlude the veins, but not the arteries. Pressure is applied for fifteen to twenty minutes and then the tourniquets are released and rotated so that one extremity is always free of pressure. The use of blood pressure cuffs instead of tourniquets presents some advantages in that the occluding pressure may be controlled. Unfortunately, three or four cuffs may not be available in an emergency situation whereas some form of tourniquet always is obtainable. If definite improvement does not occur after forty-five or sixty minutes, phlebotomy should be performed, 500 to 800 cc of blood being withdrawn rapidly. Withdrawal of less than 500 cc rarely produces the desired decrease in blood volume, but if an adequate amount is withdrawn quickly, a resultant fall in venous pressure and a rise in cardiac output can be expected. This increase in output occurs because the heart is once again able to respond to stress, i.e., the decreased venous return allows the overdistended, ineffectual

cardiac muscle fibers to decrease in size and contract more forcefully (within the limits of Starling's law of the heart). On the other hand, if the hemoglobin and hematocrit are definitely reduced, phlebotomy should not be considered. In fact, in pulmonary edema secondary to severe anemia with high output failure, small transfusions of packed red blood cells may be life-saving.

The use of Hexamethonium, a ganglionic blocking agent (10 to 50 mg. intravenously), has been advocated for the treatment of acute congestive failure as a method of reducing venous return and relieving the overdistended myocardium without actually withdrawing blood.¹⁷⁰ This drug produces vasodilatation and *peripheral* pooling of blood. There is no doubt that physiologically the use of hexamethonium is sound but its administration must be carefully controlled, and we do not believe that its routine use should be advocated for the treatment of a condition that can usually be controlled in other ways. It may be indicated, however, in patients with severe hypertension and failure.

The use of antifoaming agents in the treatment of acute pulmonary edema has not been widespread despite enthusiastic reports of some observers.¹⁰⁸ Alcohol vapor by inhalation penetrates the finer air spaces and alters the surface tension at the fluid-air interface, thereby collapsing the foam bubbles. Success with this method of therapy has been noted after all other measures have failed. Our own experience with this treatment has been limited, one of several patients so treated appeared to be helped.

Acute Cardio-pulmonary Insufficiency.—The treatment of acute right heart failure associated with chronic pulmonary disease is extremely difficult and differs in many respects from the therapy of acute left ventricular failure.¹³⁴ Chronic emphysema and/or pulmonary fibrosis increase right heart work with resultant right heart hypertrophy (*cor pulmonale*). In the presence of overactivity, infection or emotional upset, acute right ventricular failure may occur.

Severe anoxia is usually present since the degree of oxygen exchange in the lungs is markedly curtailed. The patient is cyanotic, he may be delirious or show other cerebral manifestations of anoxia and may be coughing up large amounts of thick sputum. It is extremely important to maintain adequate respiratory exchange in these persons. Morphine, codeine or the barbiturates are undesirable since they suppress respiration, but chloral hydrate or paraldehyde may be given if necessary. If oxygen is administered in high concentrations, further suppression of respiration may occur and more CO_2 accumulate (respiratory acidosis). Oxygen may be given, however, provided that adequate respiration is maintained simultaneously by the use of respirators. It may be necessary to treat the patient in this way for ten to fourteen days, utilizing the respirator ten to twenty hours a day. Hospitalization and careful supervision are necessary. Antibiotics should be given daily to combat infection, even if this is not clinically obvious. The use of vaporized bronchodilators immediately and at frequent intervals may also be beneficial (Vaponephrin—5 to 10 drops in a nebulizer inhaled ten to fifteen minutes every four hours—has proved to be satisfactory). The patient should be digitalized, given mercurial diuretics and placed on a low sodium regimen in the same manner as in left ventricular failure. Phlebotomy may be done in these patients in an effort to reduce blood volume since it is believed that the secondary polycythemic state is disadvantageous and is no longer a compensatory mechanism when the lesser circulation fails. (The hematocrit should be kept at between 45 to 50 per cent and the hemoglobin at 120 to 125 gm.) After recovery from an acute episode of cardio pulmonary insufficiency the patient should be placed on a careful regimen as outlined in a recent review on cor pulmonale.¹³⁴ The treatment of this entity is far from satisfactory, but if the patient can be successfully treated during the acute phase, in many instances he may be carried for long periods

without difficulty. The prognosis in patients with cor pulmonale secondary to pulmonary fibrosis (silicosis, berylliosis, etc.), in which there is a fixed anatomical lesion, is less favorable than in those with emphysema in whom right heart failure is more easily reversible. Acute cor pulmonale may also be caused by pulmonary infarction or overwhelming pulmonary infection without chronic lung disease and these entities should be suspected in all cases with the above clinical picture.

Therapy of Congestive Heart Failure.—After the acute attack of failure is over, the usual management of congestive heart failure is instituted. Maintenance with a suitable digitalis preparation, e.g., Digoxin, 25 to 50 mg, or gitalin, 50 to 75 mg per day (Chapter 1), a low sodium diet and the judicious use of mercurial diuretics are the three cardinal features of long term therapy. In addition, penicillin (300,000 to 600,000 u daily in a single dose) should be administered in cases of severe heart failure during the first few days when the degree of pulmonary congestion is considerable, in order to prevent secondary lung infections.⁴¹⁸ Dicumarol may be given to prevent the occurrence of phlebotrombosis and embolism, particularly in patients who will be bedridden for a long period of time.⁴¹⁷ The efficacy of this measure has been questioned by some observers, but in our experience the use of anticoagulant therapy has proved to be of value.

Adequate restriction of salt can now be achieved while the patient enjoys a reasonably well-balanced, palatable diet.⁴⁰⁶ During the first week after the acute episode of heart failure, a diet containing less than 1 gram of sodium should be prescribed. This can be accomplished by allowing only fresh fruits, unsalted vegetables, potatoes or rice, cottage cheese, salt-free bread and salt-free milk, prepared from "lanolac" powder. Many patients do not tolerate "lanolac" milk but it may be made more palatable by the use of vanilla flavoring. This preparation provides a high

protein intake with minimal sodium in contrast to regular milk which has approximately 500 mg of sodium in 1000 cc If the patient does well on this regimen, small portions of meat and fresh-water fish may be given. The patient should be warned against eating the following foods:

Salt butter, margarine and peanut butter,
 Crackers, cakes, pastries, or any food made with
 baking soda,
 Smoked or salt cured meats and fish, ham, bacon,
 pork and corned beef,
 Canned soups or vegetables,
 Pretzels, salted nuts, potato chips, candy, beets,
 celery, lima beans, spinach and sauerkraut,
 Olives, pickles, catsup, mustard and salad dress-
 ings.

A salt substitute containing potassium chloride may be used but most patients find this preparation bitter and unsatisfactory and prefer to use no salt at all. Palatability may be increased by flavoring with pepper, vinegar, garlic, onions, vanilla, chocolate, or cinnamon. Many canned foods are now available as salt-free preparations. This fact is plainly marked on the cans, and these foods may be included in the diet. There should be no restriction placed upon the ingestion of water. A fluid intake of 2 to 3 liters per day seems to be the optional intake for maximal sodium excretion^{124,162}. Tea, coffee, fruit juices, ginger ale, lemonade, salt-free milk (lanolac) and small quantities of alcoholic beverages may be taken. Frequent small meals are preferable to a few large ones, and under no circumstances should a large meal be eaten within two or three hours before bedtime. The patient should be cautioned against the use of sodium bicarbonate and hypnotics or laxatives containing sodium. If a salt-poor diet, i.e., a diet containing between 1 and 2 grams of sodium daily, is followed care-

fully, many recurrences of attacks of acute pulmonary edema or severe congestive failure can be eliminated.*

It is very important for the patient to acquire the habit of *charting his weight daily*. Only in this way can the retention of small amounts of fluid be detected. It is possible to accumulate from five to eight pounds of edema fluid before clinical signs (rales or ankle edema) appear.

Patients who are obese should be put on a fairly rigid reducing diet. We have seen patients with severe cardiac disease and intractable heart failure who were greatly improved and well controlled after marked weight reduction. Obese cardiac patients are much more difficult to control than those of average weight. Great stress, therefore, should be placed upon weight reduction as an integral part of the therapy of congestive failure. If these dietary precautions are observed, patients may be able to continue for many months without any additional therapy. If, however, the patient is unable to adhere to a proper salt-poor diet, or if his weight increases or edema develops while on such a diet, *mercurial diuretics* should be used as often as is deemed necessary to keep the patient edema-free and at a stable weight.

Intramuscular *Mercurhydrin* in 1 to 2 cc doses is a safe and effective mercurial which causes little pain at the site of injection. It may also be used *subcutaneously*. *Thiomerin*, a mercurial which is given subcutaneously, is also effective and easy to administer.^{17,127} It is possible to teach patients to administer it to themselves, as in the case of insulin administration.

Another subcutaneous mercurial preparation that we have used and found to be a satisfactory diuretic is *Dicurin* (Merethoxylline).²⁴ Its potency is about equal to the other mercurial diuretics.

*An excellent low sodium diet booklet is available through The American Heart Association for a small fee.

Since exercise increases sodium retention,¹²⁸ patients are instructed to stay in bed, if possible, for an hour or two following each mercurial injection during the early stages of therapy. In this way the maximum diuretic effect may be obtained. Occasionally minor muscle cramps occur two hours after the first dose of Thiomerin, but this discomfort disappears quickly. It is always best to administer diuretics parenterally during the initial period of treatment, and injections may be given daily for the first few days if the degree of failure is severe. Careful attention, however, must be paid to electrolyte balance in older persons.

Oral mercurial diuretics may be helpful in patients with congestive heart failure.¹²⁹ Their use had previously been limited because an effective preparation with few side effects (e.g., gastro-intestinal disturbances) was not available. At present, however, several fairly satisfactory oral preparations are available and, undoubtedly, newer more improved agents will be developed in the future. In patients who require two or more injections of a diuretic weekly it is often possible to eliminate one of these by using an oral mercurial preparation. We have used Neohydrin (chlormerodrin) frequently, and although approximately 30 per cent of patients developed signs of gastrointestinal irritation (cramps and diarrhea) and the drug had to be withdrawn, a significant number of the remaining patients were improved and required fewer injections of a mercurial. The usual dose of Neohydrin is two or three tablets daily. Recent studies have demonstrated that Neohydrin is a more effective diuretic than Cumertidin (mercuratidin), another orally effective mercurial. Usually the oral medication is not dependable enough to allow elimination of parenteral therapy, but occasionally a patient can be completely controlled by oral treatment. For example, A forty-three-year-old woman with rheumatic heart disease and mitral stenosis required the use of 2 cc of mercurhydrium intramuscularly twice weekly despite a previous mitral

commissurotomy Adequate amounts of digitalis were being given (.5 mg. Digoxin daily) but she refused to remain on a satisfactory low sodium diet Neohydrin was administered in a dose of one tablet three times daily and parenteral therapy has been unnecessary for four months No side effects have occurred in this patient

Occasionally it is necessary to use ammonium chloride, 4 to 8 gms (60 to 90 gr) a day for two or three days, prior to mercurial administration to potentiate the mercurial effect. Enteric coated tablets, containing 1 gm each, given in small and frequent doses are fairly well tolerated, but the drug should not be used continuously or in patients with marked renal impairment because of the possibility of inducing severe electrolyte disturbances (hyperchloremic acidosis). Aminophylline, 0.5 (7½ gr) given intravenously increases renal blood flow and when used one to two hours after a mercurial diuretic may markedly increase the diuretic response¹¹² Mercurials may be given in the presence of renal disease unless oliguria or azotemia is present

Great care must be taken when administering mercurial diuretics to avoid excessive diuresis which may result in salt depletion, dehydration and azotemia^{62 133} For this reason, it is wise to check the blood urea, sodium and chloride levels from time to time. When salt depletion occurs, it is often attended by serious chemical derangements and symptoms,^{133,200} the most important of which are nausea, vomiting, cramps, extreme weakness, apathy, disorientation, stupor, coma and rarely, death A decreasing urinary output despite continued mercurial therapy is often a warning sign of impending difficulty. It is extremely important to recognize and treat the electrolyte disturbances which may occur as a result of the diuretic and low sodium therapy of congestive failure Early treatment of these reactions may be lifesaving. Several types of syndromes have been noted.²⁰⁰

(1) It has been repeatedly demonstrated that more chloride than sodium is excreted as a result of mercurial therapy. If diuresis is profuse, a chloride deficit may occur, producing *hypochloremic alkalosis*. The blood sodium may not be decreased, the chlorides are usually below 85 mEq / liter and the CO_2 combining power is elevated. In this situation immediate therapy is necessary. Ammonium chloride, 4 to 8 gm., orally in divided doses or as a 1 per cent solution in glucose and water (100 cc / hour) or dilute hydrochloric acid (20 cc of U.S.P. 10 per cent acid in 600 to 1000 cc of water) may be given. (A glass tube should be used as a straw to avoid acid action on the teeth if hydrochloric acid is given.) Ammonium chloride is given routinely by many physicians (3 to 6 gm / day) during the period of rapid diuresis to prevent the above complication. It does not potentiate diuresis when used in this manner as it does when it is used prior to a mercurial injection.

(2) If excessive quantities of both sodium and chloride are excreted during rapid diuresis, a low sodium syndrome is produced. The blood sodium is usually below 125 mEq / liter and the chlorides are less than 85 mEq / liter. The blood potassium is also decreased. The use of hypertonic (5 per cent) sodium chloride intravenously with marked restriction of total fluid intake will usually correct this disturbance (80 mEq. of sodium in 100 cc 5 per cent sodium chloride). Potassium should also be given (2 to 6 gm / day as "Potassium Triplex," or solution of potassium chloride in citric acid or Karo syrup (see Chapter 1) by mouth, or intravenously (2 to 5 gm) given slowly in an infusion. If a potassium deficit persists, arrhythmias secondary to digitalis toxicity may appear^{193, 291} since lowered potassium increases myocardial sensitivity to digitalis (Chapter 1).

(3) Other electrolyte disturbances are less frequently encountered. (a) *hyperchloremic acidosis* (blood chlorides elevated, CO_2 combining power decreased), secondary to the excessive ingestion of ammonium chloride, is treated

by administering sodium bicarbonate or lactate. (b) Chronic dilution hyponatremia is accompanied by the presence of intractable edema without the symptoms noted in the "low salt syndrome." Therapy with hypertonic sodium chloride is unsuccessful and management in general is unsatisfactory.³¹⁸ The use of Southey Leach tubes or 14-gauge needles to eliminate the edema may be successful temporarily but the ultimate prognosis is poor.

While the daily injection of a mercurial is usually effective in young persons, it may be dangerous for the elderly patient. In older people only 1 cc of Mercurhydrin or Thiomerin should be given at most every other day after the acute attack of heart failure has been controlled. In elderly men, mercurials often aggravate the symptoms of prostatic disease, causing acute retention and uremia.²⁷⁸ The use of an indwelling catheter in these cases will often alleviate this difficulty and diuretic therapy may be continued. It has been demonstrated that morphine, Demerol,⁹⁸ quindine, barbiturates, ephedrine, and Adrenalin often decrease the effectiveness of the mercurial diuretics and this fact should be kept in mind if any of these drugs must be given at the same time as a mercurial.^{98 114 217}

Occasionally during the course of active therapy with diuretic agents symptoms and signs of digitalis intoxication occur in patients whose digitalis dosage had been well regulated.^{170 284} This complication of mercurial administration is not stressed, but is probably much more common than previously suspected. Although there may be some mobilization of digitalis with the edema fluid to account for this phenomenon, the urinary excretion of large amounts of potassium probably results in a sensitization of the myocardium to digitalis and in signs of digitalis toxicity. We have observed several patients whose digitalis dosage had been well regulated and who developed signs of digitalis toxicity, i.e., nausea, vomiting, and ventricular premature

beats, within twelve to twenty-four hours after the initial dose of a mercurial diuretic.

The addition of Diamox,¹⁸¹ a carbonic anhydrase inhibitor that increases the excretion of sodium, potassium and bicarbonate, to the number of oral diuretic agents presently available has helped in the management of some cases of cardiac failure. Our own experience, and that of others¹⁸² appears to indicate, however, that unresponsiveness to the drug develops rapidly even with intervals of several days between doses. While the usefulness of Diamox as an effective diuretic agent appears to be limited, it is of value in the management of patients requiring constant diuretic therapy (1 to 2 tablets, 250 to 500 mg, three or four days each week).

The judicious use of the mercurial diuretics, adequate salt restriction and digitalization usually control cardiac failure. In some patients with refractory edema, however, or in others who are not able to stay on a salt-poor diet, the use of *cation exchange resins* may be justified, if given under close supervision. The resins are substances capable of "binding" ingested sodium within the intestinal tract and excreting it in the stools.⁷⁵ The ammonium cation exchange resins with added potassium are the most satisfactory and the danger of inducing acute hypokalemia through the loss of excessive potassium in the stools is minimized with this preparation.¹⁷⁶ Adequate renal function is a prerequisite to resin therapy if severe electrolyte disturbances are to be avoided. Although the use of exchange resins *alone* does not usually eliminate edema fluid, they may help to prevent its reaccumulation by removing most of the ingested sodium. If excessive amounts of sodium are allowed in the diet, however, *even large doses* of the resins will not successfully "bind" all of it. Cation exchange resins appear to potentiate the effect of the mercurial diuretics upon urinary sodium excretion.⁸³ Electrolyte disturbances are not frequent when the ammonium-potassium resin is used and

when careful attention is paid to blood electrolyte levels. Constipation may prove to be an annoying symptom since large quantities of resin material must be ingested to produce an effect (30 to 45 gm. to eliminate approximately 1.5 to 2.0 gm. of sodium). Our experience with the exchange resins suggests that they may be a useful adjunct in the therapy of congestive heart failure only in selected cases.

In addition to the above measures, the patient's activity should be carefully regulated. Short periods of bed rest should be taken if symptoms become severe or if heart failure appears to be progressing.

In heart failure secondary to hyperthyroidism, pernicious anemia, or nutritional deficiencies, the correction of the primary disease usually prevents its recurrence. In cardiac failure secondary to tachycardia treatment of the latter condition should be instituted, as outlined above. Unusual etiologies of congestive failure should be eliminated before a case is labeled "refractory" to all therapy. An interesting example of this was a twenty-eight-year-old man who for eight years had been treated for increasingly severe heart failure. Cardiac enlargement was present but no evidence of rheumatic, congenital, nutritional or metabolic heart involvement could be discovered. A careful search led to the discovery of an arteriovenous aneurysm in the right buttock area (the result of a World War II shrapnel wound). Repair of the aneurysm led to a complete cure of his "refractory" heart failure.

In patients with heart failure, fluid may accumulate rapidly in the chest and cause acute distress. In these cases immediate and repeated thoracenteses give remarkable relief and permit recovery. Accumulation of fluid in the abdominal cavity also occurs but rarely does enough fluid collect to cause symptoms and require paracentesis. This has been especially true in recent years since the use of palatable salt-poor diets and adequate mercurial therapy became widespread. Occasionally, in patients with severe

rheumatic heart disease and long-standing congestive failure, massive ascites develops as a result of advanced cardiac cirrhosis, we have observed patients who required frequent paracenteses in order to remain comfortable. One of these was a thirty-four-year-old female who was hospitalized because of chronic congestive heart failure secondary to rheumatic heart disease. Bilateral pleural effusions and ascites were found. Despite adequate digitalization, marked salt restriction and the frequent use of mercurial diuretics, little improvement was noted after one week of therapy. A paracentesis was performed and 7 liters of fluid were removed. This was followed by a marked diuresis with dramatic clinical improvement. Another paracentesis was done one week later; thereafter, the patient was controlled without "tapping."

Cardiac cirrhosis and liver disease in patients with chronic congestive failure are much more common than previously suspected,^{30, 163} and for this reason attention should be paid to the patient's intake of protein and vitamins. Because these patients are often on a strict diet, there is a tendency to overlook simple measures such as the use of protein and vitamin supplements that might postpone the occurrence of severe liver disease and ascites.

In extremely refractory cases of congestive heart failure the induction of hypothyroidism by the use of radioactive iodine is occasionally successful. Another procedure that has been advocated for the treatment of refractory failure is ligation of the inferior vena cava.^{63, 170} The operation has a definite morbidity and mortality, is merely palliative and the long term results are equivocal.

During attacks of acute heart failure, especially following coronary occlusion, hiccup, severe nausea, vomiting, and distention may develop and produce serious difficulties. It is of great importance to prevent these symptoms or, if they occur, to treat them quickly.

The excessive use of opiates should be avoided if possible, and laxatives should be given freely to avoid constipation.

If frequent narcotics are being given, two and even three times the usual dose of a laxative may be necessary. If therapy with laxatives such as Milk of Magnesia (30 to 60 cc.) or *Cascara sagrada* (10 to 30 gr.) is not successful, occasional low gentle enemas should also be given. We have found the "Fleet's" enema preparation especially useful in these cases. Cathartics such as rhubarb that act upon the large bowel should not be used in patients receiving mercurials because of the possibility of inducing colitis.

The use of Dramamine and Thorazine*¹¹ (chlorpromazine hydrochloride, SKF) have greatly facilitated the treatment of the nausea and vomiting that complicate congestive heart failure. Dramamine may be given orally, rectally or intramuscularly in a dose of 50 mg. Thorazine is given intramuscularly (10 to 25 mg) for the first dose and then orally (10 to 25 mg every six hours) until symptoms are no longer present. It is a most effective antiemetic agent. Transient postural hypotension and tachycardia may occasionally be noted after the first few doses. We have had great success with this drug. The sedative effect of Thorazine may also eliminate the necessity of using sedatives and narcotics while it is being given. (Thorazine potentiates the effect of these other agents if they are given simultaneously.)

Nausea and vomiting may occur as a result of overdigitalization or following the use of morphine, aminophylline or ammonium chloride. In these instances, the offending agent should be withdrawn.

The therapy of intractable hiccup is extremely difficult. Many drugs and maneuvers have been tried, each producing success at one time and failure at others. Sedation, amyl nitrite, intravenous niacin, inhalation of 5 to 7 per cent CO₂ and 90 per cent oxygen for a few minutes at a time, ethyl chloride spray to the region over the diaphragm,

*Chlorpromazine hydrochloride "Largactil" in Canada, England, France and Italy; "Megaphen" in Germany, "Amplijactil" in Argentina.

inhalation of ether, or intramuscular quinine, gr 6 (4 gm) three times a day³⁸ should all be tried in turn in the hope that one of them will be successful in stopping this debilitating symptom. Thorazine in large doses (25 mg every four hours) may also be effective in some cases, perhaps largely due to its sedative effect. A phrenicectomy or exeresis is occasionally required to stop a severe attack.

Chapter

3

ANGINA PECTORIS, CORONARY INSUFFICIENCY AND CORONARY OCCLUSION

TREATMENT

THE most common cardiac emergency is pain in the chest. First, it is essential to exclude extracardiac causes of chest pain such as spondylitis, gallbladder disease, peptic ulcer, hiatus hernia, esophageal spasm, herpes zoster, pneumothorax and pleurisy, before deciding that the pain is cardiac in origin. This can usually be done by taking a careful history and doing a complete physical examination, but an electrocardiographic study may be necessary.

Once the cardiac origin of the pain has been established, the type of acute cardiac episode must be determined. The character, intensity and duration of the pain may help the physician to distinguish between attacks of *coronary insufficiency* (in which there is no acute closure) including *angina pectoris*, and *coronary thrombosis or occlusion*. In coronary occlusion the pain is usually prolonged, severe and unrelieved by nitrates, whereas, in coronary insufficiency, particularly *angina pectoris*, it is briefer and is relieved by nitroglycerin. Occasionally, however, coronary occlusion is associated with only mild pain and no other symptoms, whereas coronary insufficiency may cause severe pain. The entire clinical picture and the electrocardiographic changes should be considered before a definite diagnosis is made, and this frequently is possible only after observation for

several days. Any patient, therefore, who experiences sudden precordial pain, diagnosed to be cardiac in origin, should be put at rest and treated as a case of potential infarction, although many of these cases do not progress to a coronary occlusion with infarction.

✓**Angina Pectoris.**—Angina pectoris is usually precipitated by effort, exposure to cold, excitement or large meals and represents temporary acute coronary insufficiency, with ischemia of the subendocardial layer but no myocardial necrosis. The ordinary episode of angina pectoris is quickly relieved by rest or nitroglycerin although the drug is not effective in over 10 per cent of cases. A hypodermic tablet, gr 1/200 to 1/150, is taken under the tongue. The patient should be instructed to take the nitroglycerin as soon as the pain appears. If a specific activity, such as eating or walking in cold weather, is known to induce anginal pain, the drug should be taken prior to this activity. For example, there are many patients who experience pain only in the morning on the way to the subway or train, a nitroglycerin tablet, placed under the tongue just before leaving the house often prevents the attacks. This drug is safe and extremely effective, and should be taken as often as necessary. There is no danger in giving nitroglycerin as frequently as every hour or even less. The patient should be warned, however, not to take more than two tablets within five or ten minutes. Additional tablets will probably not give relief and are apt to produce discomfort. If the drug is not effective, one should suspect coronary occlusion.

Occasionally a single nitroglycerin tablet produces flushing, intense throbbing and severe headaches, or even syncope, and, in these instances, a minute dose, gr 1/400 or less, should be used. Even this dose may cause symptoms. For example, A forty-eight-year-old waiter suddenly experienced substernal pain and was given gr 1/150 of nitroglycerin. Severe throbbing in the head, which lasted several hours, profuse perspiration and "faintness" occurred,

but the pain was not relieved. During another attack of precordial pain, several days later, the patient was prevailed upon to try nitroglycerin again in a minute dose of gr. 1/400, but the same symptoms were produced.

On rare occasions we have prescribed less than 1/400 gr. to avoid a reaction. While such a small dose may relieve pain, it is apt to be ineffective. Nevertheless, it is worth trying in a patient who can not tolerate a larger dose. Also, a patient may react badly to nitroglycerin at one time but respond well at a later date.

Some patients are averse to taking nitroglycerin because they have heard that it is dangerous or habit forming. It is essential to convince the patient that these notions are false and that the drug is not only effective but safe. It is important psychologically that the patient take nitroglycerin, if it is not used, the pain tends to persist and the patient becomes frightened and anxious. This state is apt to perpetuate the anginal syndrome by establishing a vicious cycle of pain, anxiety and more pain, often resulting in "status anginosus."

While nitroglycerin relieves anginal pain and can prevent it if it is taken immediately before a specific activity is undertaken, it is not effective in the general prevention of anginal attacks. For this purpose we have found reassurance, frankness and optimism about coronary disease, some restriction of activity if necessary, loss of weight, eradication of irritating foci and sedation valuable. Over the years many vasodilator drugs, particularly the xanthine group, have been used to prevent anginal pain without success. The latest drugs used, none of which has proven to be truly effective, include:

- (a) PERITRATE (Pentaerythritol tetranitrate). A long-acting nitrate, has been found to prevent the appearance of coronary insufficiency following a 2-step exercise test,²⁷² but clinical studies have produced conflicting results and our experience has been disappointing.

At best, it appears to be effective only in an occasional patient in a dose of 10 mg four times a day

- (b) METAMINE (Triethanolamine trinitrate biphosphate) Another long-acting nitrate, has proven of little value. The dose is 2 mg four times daily.²²⁰
- (c) NITROGLYN A sustained action nitroglycerin tablet (gr 1/25 and gr 1/10) is said to allow release of the drug gradually over a period of ten to twelve hours. As yet there are no published reports concerning this drug, but it has not been successful in our experience.
- (d) KIELLIN An extract from the seeds of an Egyptian plant, dilates the coronary arteries when given intramuscularly in a dose of 100 mg., or orally in 40 to 60 mg. doses three times a day. Early favorable reports on this drug have not been confirmed^{126, 241} and we have found that it usually produces nausea and vomiting in ordinary doses without relieving anginal pain.
- (e) PAPAVERIN DERIVATIVES Papaverin has been shown to be ineffective in the anginal syndrome in doses up to 800 mg. daily.²⁴² A derivative, *Paveril Phosphate*, was recently introduced with an optimistic report but our own experience with this drug has been disappointing.²²⁰
- (f) HEPARIN Heparin has been given intramuscularly or intravenously two or three times a week, in doses of 50 to 100 mg. for anginal pain. Although good results were reported initially, subsequent studies, adequately controlled, have shown the drug to be ineffective.²²⁰
- (g) QUINIDINE, TESTOSTERONE, VITAMINE E, (ALPHA TOCOPHEROL), HYDERGINE, NICOTINIC ACID, and AMINO-OPHYLLINE-ALUMINUM HYDROXIDE MIXTURES These have been used in angina pectoris, but without effect.
- (h) RESERPINE (*Rauwolfia serpentina*) Occasionally effective in treating hypertension, has been used by us in

tense patients with angina pectoris without hypertension, and has appeared to help an occasional patient. At times a patient experiences less pain with only one tablet a day.

- (i) **ALCOHOL.** Although there is a prevalent notion that alcohol is beneficial in coronary disease and angina pectoris, there is no evidence that it produces dilatation of the coronary arteries.²⁷⁵ Occasionally alcohol relieves acute anginal pain when nitroglycerin is unavailable or ineffective. This effect is probably associated with the relaxation produced.

Status Anginosus and Angina Decubitus. During the course of a chronic anginal syndrome, there may be a sudden exacerbation of recurrent pain occurring at rest and at night, as well as on effort. This chain of events, known as "status anginosus," may follow an emotional upset and is usually a serious situation because the patient becomes very apprehensive as a result of the recurrent severe pain. It is important to allay the patient's apprehension by reassurance, by using a strong sedative or a narcotic such as Dilaudid, gr 1/32 or 1/48 three or four times a day for seven to ten days, and by the liberal use of nitroglycerin for the pain. Nitroglycerin usually relieves pain during this period of "status anginosus" which indicates that acute coronary insufficiency has occurred or that a coronary occlusion is forming or has formed. If nitroglycerin becomes ineffective, coronary occlusion with major infarction has probably developed and the drug should be stopped immediately. During this period the patient should be at complete rest in a chair in his room.

While the period of "status anginosus" usually subsides after one to four weeks, occasionally the pain continues to recur frequently. Also, occasional patients with a chronic anginal syndrome may fail to improve and continue to be unable to walk more than a short distance. In such refractory patients, three types of therapeutic procedures are

available to the physician. (a) lowering the metabolism with radioactive iodine (I_{131}), (b) interrupting the cardiac nervous pathways, (c) performing an operation to increase coronary flow and induce extracardiac collateral circulation

(a) RADIOACTIVE IODINE (I_{131})

This drug, which has largely replaced thyroidectomy³¹ and antithyroid drugs,³⁶⁷ is, of course, very efficacious in treating angina pectoris secondary to hyperthyroidism, but it may also be effective in relieving intractable angina in euthyroid patients.³⁸ In the latter, a hypothyroid state is induced but severe hypothyroidism may be prevented by careful regulation of the dose. If severe hypothyroidism develops, it can be relieved by administering small doses of thyroid.

According to some observers,³⁸ more than 50 per cent of patients thus treated are relieved of anginal pain. The method is quite safe and may be tried in patients who do not respond to the usual methods of treating angina. It must be remembered that the effect of I_{131} does not appear for at least one or two months after its administration. If I_{131} is unavailable, propyl thiouracil may be employed using necessary precautions.

A dramatic example of the effect of I_{131} in a euthyroid patient was a forty-six-year-old woman who developed an attack of acute coronary insufficiency with T-wave inversions. She recovered completely and the electrocardiogram returned to normal. She was free of pain for five years and then experienced frequent severe angina at rest and during the night. She required 10 to 20 nitroglycerin tablets daily. At this time her electrocardiogram showed T-wave inversion and on several occasions the inversion became deeper for seven to ten days. Radioactive iodine tracer studies showed her to be euthyroid. She received a therapeutic dose of I_{131} . She continued to experience severe pain during the next seven weeks and since then has been free of

pain. It is very likely that the improvement in this patient was the result of the I_{131} and was not spontaneous.

It is important to exclude the presence of actual hyperthyroidism in patients with severe angina pectoris, for only correction of the hypermetabolic state relieves the pain, as the following case report demonstrates.

A.F. was a sixty-seven-year-old Italian housewife who entered the hospital because of severe, recurrent episodes of substernal pain which began suddenly two weeks before. Her blood pressure was 160/80, her rhythm was regular, and a grade ++ apical systolic murmur was heard. The electrocardiogram showed deeply inverted T-waves in the standard and precordial leads, a diagnosis of coronary insufficiency was made. In spite of complete bed rest and large doses of sedatives, she continued to experience frequent attacks of pain which were only partially relieved by nitroglycerin. During the course of a thorough study her basal metabolic rate was found to be +40 per cent, and she excreted only 10 per cent of a test dose of I_{131} , indicating hyperthyroidism. She was given a therapeutic dose of I_{131} and after several days, Lugol's solution. Her anginal seizures began to subside after several weeks and finally disappeared completely.

(b) INTERRUPTION OF CARDIAC NERVOUS PATHWAYS AND IMPULSES

These procedures include, in the order of increasing seriousness (1) *paravertebral alcohol block* of the upper five thoracic sympathetic ganglia bilaterally, (2) excision of these ganglia (*thoracic sympathectomy*) and (3) laminectomy with section of the upper five dorsal roots (*posterior rhizotomy*).³¹⁰⁻³³⁴ Sympathectomy appears to be the most effective of these and the operation is well tolerated.³⁰

(c) INDUCTION OF COLLATERAL CIRCULATION TO INCREASE CORONARY FLOW

A number of operative procedures devised years ago to induce collateral circulation between the heart and sur-

rounding tissues have been discarded,⁶⁴ however, the implantation of a substance such as talcum into the pericardial cavity (cardiopericardioplexy) is again in vogue in some quarters.¹¹³ An adhesive pericarditis is produced and although the degree of collateral circulation developed is a moot question,⁴⁶ symptomatic improvement has been reported in 50 per cent of patients.¹¹⁴ The operation is simple and the mortality rate is said to be low. Therefore, in the occasional patient with intractable angina who has not responded to radioiodine, the operation seems worthy of trial.

Three other surgical procedures have been performed to increase coronary flow but must still be considered experimental and of unproven value. They are

- (1) Cardiac vein ligation to increase the blood flow through the Thebesian vessels.⁹³
- (2) Arterialization of the coronary venous system by anastomosing the aorta to the coronary sinus by means of a venous graft.¹⁰
- (3) Arterialization of the intramural arteries by implantation of an internal mammary artery into the anterior wall of the left ventricle.³²⁰

It must be remembered that many patients with severe anginal pain, or even "status anginosus" which persists for many weeks or months, may eventually become symptom free on a good medical regimen. Operation for angina pectoris should be considered only after all other therapy has failed and the effect of such a procedure has been carefully evaluated.

Coronary Insufficiency.—Many acute coronary episodes are more prolonged than the transitory attack of angina of effort but less severe than a typical acute coronary occlusion. We have suggested that the name acute coronary insufficiency without thrombosis be restricted to these attacks.^{212,213} Although acute coronary insufficiency may resemble a mild coronary occlusion clinically they differ

84 Angina Pectoris, Coronary Insufficiency

electrocardiographically and pathologically. In coronary insufficiency the electrocardiogram shows RS-T depressions and/or T-wave inversion in any leads (Fig 7), Q waves and RS-T elevation, which characterize coronary occlusion, are absent. Acute coronary insufficiency may result in: (1) ischemia of the subendocardial layer, in which case the

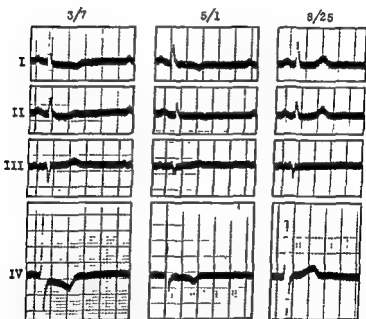


FIG 7—N.C., m 53. Acute coronary insufficiency following physical exertion. (Master and Jaffe, courtesy of Postgraduate Medicine.)

electrocardiographic abnormalities disappear within several days, or (2) subendocardial necrosis, in which case the electrocardiographic changes may persist for weeks, and mild fever and elevation of the sedimentation rate may develop.

Acute coronary insufficiency represents a discrepancy between the supply and demand of coronary flow. It may

occur spontaneously or may be induced by conditions which increase the work of the heart, diminish the coronary flow or interfere with oxygenation of the blood. These include effort, emotion, massive hemorrhage,²¹⁴ paroxysmal tachycardia, operative procedures or other conditions associated with shock or a drop in blood pressure (e.g., intravenous pyclography or the use of anti-hypertensive drugs), acute infections such as gastro-enteritis, acute abdominal conditions such as cholecystitis and pancreatitis, hypertensive crises, pulmonary embolism and infarction, aortic stenosis and heart failure.

- **Spontaneous Coronary Insufficiency.**—The majority of spontaneous attacks of coronary insufficiency pursue a mild course and the prognosis is excellent. In other cases, however, the attack represents the *premonitory phase of coronary occlusion* and the pain may recur for several days or weeks before the acute occlusion occurs.¹⁵⁰ Careful observation is necessary with the patient at rest in a chair for one or two weeks. If it is suspected that a coronary occlusion is forming, anticoagulants may be administered in order to prevent its completion but our results have been disappointing in this respect. It is impossible to predict which cases will recover and which will go on to complete occlusion. For example, Mr H began to experience repeated anginal pain at night and during the day. His electrocardiogram showed mild T-wave changes. He was given nitroglycerin and Dilaudid, his pain disappeared after several days and his electrocardiogram returned to normal. This was a case of acute coronary insufficiency with spontaneous recovery.

On the other hand, Mr S, who gave an identical history, was given Dicumarol but developed a coronary occlusion one week later.

The benign prognosis of spontaneous acute coronary insufficiency is illustrated by the following cases

Mr. I.F., a storekeeper, forty-four-years-old, had been observed over a number of years because of numerous functional complaints. Physical examination, blood pressure, and electrocardiogram were normal. One day, while at work, he experienced severe substernal pain which lasted one hour and suggested the diagnosis of coronary occlusion. He was not in shock, his heart sounds were regular and of good quality at a rate of 76. There were diphasic T-waves in leads I, II, and V_4 , but his temperature was not elevated and there was no leucocytosis. He remained asymptomatic but on the fourth day his sedimentation rate was elevated to 30 mm and his electrocardiogram showed inverted T-waves in all leads, without Q-waves. The T-waves gradually became upright and in four weeks the electrocardiogram was again normal. He remained well during the next three years and his electrocardiogram was unchanged after exercise. *In this case the acute coronary insufficiency caused some subendocardial necrosis but recovery was complete.*

A J., a sixty-three-year-old automobile salesman, with a history of bronchial asthma for many years, complained of sudden, severe pain across the upper back radiating anteriorly. He perspired freely, his blood pressure was 130/80, a drop of 30 points from his usual systolic pressure. The pain lasted forty-five minutes and was then relieved by 100 mg of Demerol subcutaneously. His electrocardiogram was normal and, after four days he was permitted in a chair. He again began to experience frequent, severe pain in the upper back during the day and particularly at night. Because an incipient coronary occlusion was suspected, Dicumarol therapy was begun and the patient received oxygen by mask, aminophyllin suppositories, Demerol 100 mg every four hours and sedatives. The pain increased in severity but the patient's temperature, white count and sedimentation rate remained normal. On the eleventh day the electrocardiogram showed slight inversion of the T-

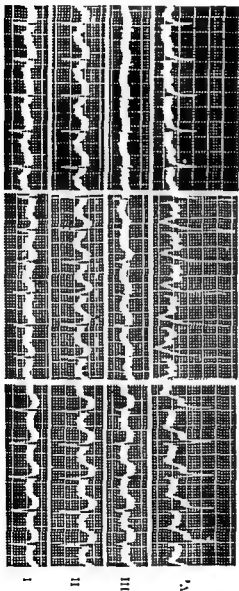


Fig. 8—P1. in 36 Acute coronary insufficiency in duodenal ulcer with profuse hemorrhage. Necropsy showed subendocardial necrosis. A, Marked RS-T depression in all leads. B, RS-T depression in all leads. C, RS-T depression less marked. C, further improvement (Master, Dack, Horn, Freedman and Field, courtesy of Circulation)

waves in leads I and V_2 to V_3 , but no other changes. The T-wave inversion gradually increased during the following two weeks and then receded, four weeks after the acute episode the electrocardiogram had returned to normal. *The final diagnosis was therefore acute coronary insufficiency. Although the temperature and sedimentation rate remained normal, subendocardial necrosis was doubtless present since the T-wave changes persisted for several weeks.*

Coronary Insufficiency Secondary to Other Conditions.—One of the most frequent and important causes of acute coronary insufficiency is gastro-intestinal hemorrhage, particularly when there is a marked fall in blood pressure²¹⁴ (Fig 8). Prompt recognition and treatment are essential if the patient is to recover. Repeated transfusions may be necessary and we have not hesitated to give up to 2500 cc of blood in twenty-four hours, in order to keep the blood volume and blood pressure as close to normal levels as possible. As a rule these patients tolerate large amounts of blood well, should evidence of pulmonary congestion or edema appear, a mercurial diuretic is usually effective and digitalis should be given. The progress of the coronary insufficiency can be followed by serial electrocardiograms. As improvement occurs, the T-wave inversions disappear. In the occasional fatal case, necrosis of the subendocardial layer is found but there is no evidence of acute thrombosis¹⁰⁰.

Every effort to prevent attacks of severe coronary insufficiency should be made in patients with evidence of coronary disease. They should be advised not to indulge in strenuous activities, they should also be warned against excessive eating, exposure to extreme changes in temperature, frequent sexual intercourse, emotional upsets and excessive smoking. It is important to prevent a fall in blood pressure, anoxia, tachycardia or hemorrhage when such patients are subjected to operation. If spinal anesthesia is used the blood pressure should be watched carefully and

neosynephrin given if the systolic pressure falls below 100 mm Hg. If considerable bleeding occurs, blood for a transfusion should be readily available.

In diabetic patients with coronary disease who are receiving insulin it is important to prevent episodes of hypoglycemia, it is better to allow them to spill small amounts of sugar than to attempt to attain perfect sugar control, since hypoglycemic reactions may precipitate episodes of coronary insufficiency. If the above precautions are followed many cases of this syndrome will be prevented.

Coronary Thrombosis or Occlusion.—In coronary thrombosis a fresh thrombus obstructs the lumen of one of the major coronary arteries. The pain experienced in coronary occlusion usually begins suddenly, at rest, during sleep or in the course of routine activity. It is usually precordial or substernal, but may be situated only in the back of the chest or neck, or in the arms. Instead of pain, the patient may complain of severe burning sensations or feelings of tightness, nausea, vomiting, and profuse perspiration. Varying degrees of shock may be present. The sudden onset of paroxysmal tachycardia or pulmonary edema is occasionally the first evidence of an acute coronary occlusion. Examination may reveal gallop rhythm, embryocardia, (tic-tac rhythm), or congestion of the lungs. The blood pressure may be normal at first or it may drop precipitously.

The clinical and electrocardiographic manifestations of coronary occlusion depend upon the pathological changes that occur in the coronary arteries and myocardium. In over half the cases the process of thrombosis is initiated by a subintimal hemorrhage which leads to secondary changes in the intima or completely occludes the vessel lumen.¹⁴⁷ This process of thrombus formation may take days or weeks. During this period the patient may experience severe episodes of angina or recurrent pain at rest, the so-called "premonitory phase" of an occlusion.¹²⁸ The electrocardiogram may show RS-T depressions and T-wave inversions, indi-

cating coronary insufficiency with myocardial ischemia. These changes are replaced by Q waves and ST segment elevations, however, as soon as the occlusion has become complete (Fig 9) In coronary occlusion a Q-wave usually

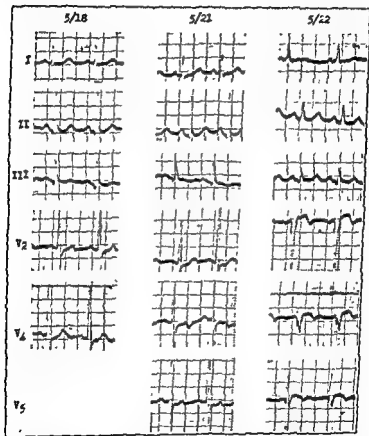


FIG 9—NH, m 45 RS-T depression and T-wave inversion in the chest leads, indicating coronary insufficiency, during the premonitory phase of coronary occlusion (5/18, 21) Following the acute attack RS-T elevation and Q-waves appeared in leads V₂ and V₄ (5/22), indicating anterior infarction (Jaffe, courtesy of J Mt. Sinai Hosp)

appears in one or more leads, depending upon the site of the infarction, and produces a characteristic pattern of a Q-wave and RS-T elevation (Fig 10). During the following days and weeks, serial tracings reveal a progressive return of the RS-T segment to normal, and an inversion of the T-wave, resulting in a Q, inverted T pattern which may persist for months or years. Fever appears on the second or third day, the sedimentation rate becomes prolonged by the fourth day, often to 50 mm or more. Since the infarction is "through and through," the subpericardial region is

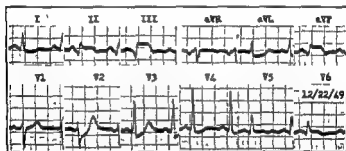


FIG 10—J S, m 58. Acute coronary occlusion with postero-lateral infarction (Master and Jaffe, courtesy of Postgraduate Medicine)

involved, and a pericardial reaction²⁰⁸ and friction rub may result. The infarct also reaches the endocardium, therefore mural thrombi may form and peripheral emboli develop.¹³⁸ In coronary insufficiency without thrombosis the necrosis is limited to the subendocardial area and only RS-T depression and/or T-wave changes occur.

At times it is difficult to distinguish between a coronary occlusion and acute pericarditis. Some of the features that differentiate these conditions from one another and from coronary insufficiency are summarized in Table 5. The most reliable distinguishing feature is the presence of Q waves in the electrocardiogram, which are characteristic of

coronary occlusion. It is important not to mistake pericarditis for coronary occlusion since anticoagulant therapy is contraindicated in acute pericarditis, for the reason that hemorrhagic effusion may occur.

In treating coronary occlusion, it is essential to relieve the pain as soon as possible. If it is not very severe, Demerol,

Table 5. Differential Diagnosis of Acute Coronary

	Pain	Severe Sweat	Gallop Rhythm	Peri- cardial Rr's	Heart Failure	Arrhyth- mia	Drop in Blood Pressure
A Coronary Insufficiency							
a) Mild— Angina Pectoris	Few seconds to 10-15 min- utes	Absent	Usually ab- sent	Absent	Absent	Absent	Absent
b) More Se- vere— spontane- ous or Secondary	May be pro- longed	May be pre- cipitating factor, e.g. hemorrhage	May occur	Absent	Uncommon except in cases due to tachycar- dia	Uncommon	Moderate if ep- isode precip- itated by hemorrhage or tachycar- dia
B Coronary Occlusion	Usually severe longer than 15 minutes, rarely absent	Present in about 10 per cent of cases	Common	Not un- common Usually transitory Often localized	Common	At 15 per cent	Often marked
C Pericarditis	Mild, occasion- ally severe Often related to respiration	Rare	Rare	Usually pre- sent Often widespread Often per- sistent	Uncommon	Atrial flutter and fi- brillation not un- common	Rare unless cardiac tam- ponade occurs

50 to 100 mg subcutaneously, usually suffices. For more severe pain, morphine, 15 mg (gr. 1/4) subcutaneously or 10 to 15 mg. (gr. 1/6 to 1/4) intravenously, may be required. The intravenous route is the more effective. The drug may be repeated in the same doses in twenty to thirty minutes. The possibility of inducing distention, obstipation, nausea and vomiting with morphine is great and the dose used should not be larger than is actually required.

Morphine should be discontinued if any of the above symptoms appear since distention and vomiting may turn an otherwise benign course into a stormy one. If the patient is receiving oxygen there is little danger of respiratory depression. We have used Dilaudid extensively and have found it to be a reliable analgesic which produces fewer

Insufficiency, Coronary Occlusion and Pericarditis

TYPE	LEUKOCYTES	ELEVATED SEDIMENTATION RATE	ECG R-S-T E1 R-S-T Dep Q Waves	PATHOLOGY	PRECIPITATING FACTOR	TREATMENT
Absent	Absent	Rare	R-S-T E1 R-S-T Dep Transitory Q-Waves Absent	No pathological changes	Emotion, exertion, cold large meals, etc.	Nitroglycerin in status anginosus-paroxysmal (for short period)
May be present	May be present 10,000-15,000	Mild to Moderate	(R-S-T E1 R-S-T Dep Common Q-Waves Absent)	Subendocardial necrosis often present	1) Tachycardia, hemorrhage, shock, trauma and as in status pectoris 2) Spontaneous	1) Treat etiological factor 2) Symptomatic
Usually present after 24 hours May be high	Usually present after 24 hours, may be over 15,000	Usually after 72 hours Often very high	R-S-T E1 (R-S-T Dep Reciprocal) Q-Waves Common	Through and through infarction of myocardium	Shock (except shock ¹)	Symptomatic anticonvulsants (see text)
Common at onset	Depends on etiology If viral leucopenia	Common at onset	R-S-T E1 Common (R-S-T Dep Rare) Q-Waves Absent	No myocardial damage (may be subepicardial)	Nonspecific (viral?) rheumatic fever bacterial traumatic tuberculous, other diseases	Treat primary disease

unpleasant and dangerous side effects than morphine. Two and one-half to 5 mg (1/24 to 1/12 gr) usually relieves pain. We have also found Pantopon and Metapon satisfactory. Nitroglycerin does not relieve the pain of acute coronary occlusion and should not be used since it may further lower blood pressure and decrease coronary blood flow.

Oxygen should be given by tent or mask when the pain

94 Angina Pectoris, Coronary Insufficiency

persists after morphine has been given. A tent usually induces less apprehension than does a mask. Occasionally, intravenous aminophylline 5 gm. (75 gr.) is of value. It should be injected slowly. In rare instances, sodium amytal, .5 gm. (75 gr.) by vein has been effective when all other measures have failed.

As soon as the pain has been controlled, the question of hospital or home care arises. The vast majority of patients can be cared for at home, unless conditions there are unsatisfactory. Visitors other than the immediate family and the use of the telephone should be banned. Patients with a coronary thrombosis are apprehensive and discouraged. It is extremely important to encourage them, stating frankly that they have had a coronary attack but that most patients make a complete or satisfactory recovery and lead full lives.²¹⁹

The most serious complication of coronary occlusion is *shock* which in the past was almost always fatal.

The mechanism of "*cardiogenic shock*" is not entirely clear but it has been shown that in acute myocardial infarction there is a reduction in cardiac output²¹⁴ and stroke volume and severe, generalized vasoconstriction.¹⁰³ This may produce not only a state of shock but also fluid retention and heart failure. Theoretically the use of transfusions and intravenous fluids is not indicated and, in fact, has proven of little value although they only rarely induce heart failure.^{88, 216} Intra-arterial transfusions have also been found to be ineffective.²⁰

The outlook in "*cardiogenic shock*" in the past few years has considerably improved as the result of the use of vaso-pressor drugs. When these are administered *within the first few hours*, occasional patients have recovered. The drugs used include: (1) Norepinephrin (Levophed)²¹⁷ This must be given slowly by the intravenous route (4 mg. in 1000 cc. glucose in water). The infusion should run rapidly enough to maintain the systolic pressure at approximately 100 to

110 mm Hg, avoiding higher levels. The drug is continued as long as the pressure tends to fall without it. Levophed is safe if its effect is carefully observed. Its major effect is on the blood pressure although it may increase cardiac irritability to some extent and rarely cause arrhythmias. Localized superficial gangrene, as a result of intense vasoconstriction, has been observed in several patients who had received Levophed for long periods. The drug often exerts a rapid and marked effect, as the following case demonstrates. Mr. D. developed a coronary occlusion and diaphragmatic infarction and his blood pressure did not rise above 90/70 mm for five days. A Levophed intravenous infusion was then started with 4 mg per liter, forty-five minutes later his blood pressure was 170/120. The rate of flow was then slowed as much as possible in order to keep the systolic blood pressure below 120. (2) Mephentermine (Wyamine). The dose is 10 to 20 mg intravenously, or 15 to 35 mg, intramuscularly repeated as often as indicated (every thirty to forty-five minutes). Wyamine does not affect myocardial irritability.⁴¹ (3) Phenylephrine (Neosynephrin)⁴² 1 mg intravenously or 5 to 10 mg intramuscularly, repeated every hour or two, if necessary, and (4) Methoxamine (Vasoxyl),²⁴⁴ 20 mg intravenously or intramuscularly, have also been used with success.

The use of these drugs requires constant observation but the time spent will be amply repaid if even a small percentage of patients recover from this very serious condition. One of these drugs should be in the doctor's bag, for the sooner the treatment is instituted the better the outlook.

A patient in shock should immediately be placed in a moderate Trendelenburg position and oxygen administered if available. The patient is usually apprehensive and, particularly if pain is present, a narcotic should be administered. In those cases with pulmonary congestion or other evidence of congestive failure, intravenous Strophanthin or Cedilanid should be administered as previously described⁴⁰ (Chapter

1) In some instances clinical signs of heart failure may be absent, yet the venous pressure, if determined, is found to be elevated. Consequently, if a patient in shock does not respond to vasopressor drugs and the other measures described above, it is worthwhile to administer digitalis for occasionally the result is gratifying.

Clinical evidence of mild *left ventricular failure* is very commonly observed early in the course of coronary occlusion, a few rales are found in the chest and slight congestion of the lung fields is seen on x-ray. As a rule, this type of failure requires no specific therapy but responds promptly to moderate salt restriction and to an injection of a mercurial diuretic. Not infrequently, however, acute pulmonary edema is the first or only sign of a coronary occlusion, or severe heart failure develops within several days after the acute episode. As an example of the former, we observed a woman of sixty-four who suddenly developed attacks of severe pulmonary edema but who had experienced no chest pain whatsoever. Nevertheless, the electrocardiogram revealed evidence of an acute coronary occlusion with myocardial infarction. Doubtless, this had occurred at the time of the first attack of pulmonary edema.

If pulmonary edema occurs, the treatment is as outlined previously for *pulmonary edema* in the absence of coronary occlusion (Chapter 2), i.e., morphine or another narcotic and, if necessary, intravenous aminophylline, intravenous Strophanthin or Cedilanid, oxygen, a mercurial diuretic, and phlebotomy unless a severe state of shock exists.

The treatment of *congestive failure* in coronary occlusion is the same as in chronic heart disease although there is some difference of opinion concerning the use of digitalis. Many physicians believe that it should be used in coronary occlusion whenever heart failure is found,¹⁰⁵ but some, including one of the present authors, prefer to use diuretics and a low salt diet before administering digitalis and, if necessary, to digitalize somewhat more slowly than usually.

This does not hold for an acute emergency like pulmonary edema. Caution in the use of digitalis in coronary occlusion is exercised because of the potential danger of inducing *arrhythmias*, particularly *ventricular tachycardia*.

Arrhythmias of all types occur frequently in coronary occlusion. The most common are *premature beats* and *paroxysmal atrial fibrillation, flutter and tachycardia*. They do not appear to alter the prognosis very significantly since they often are of brief duration and remit spontaneously. For this reason it is not necessary to begin specific treatment of these *arrhythmias* immediately if the patient is comfortable. Sedatives should be used liberally because the tachycardia is apt to cause anxiety. If the *arrhythmia* does not cease within one-half to one hour, specific therapy should be instituted as outlined in Chapter 1. If there is any indication of incipient heart failure or shock at the onset of the *arrhythmia*, treatment should be started promptly.

The temporary nature and spontaneous remission of paroxysmal tachycardia in coronary occlusion is illustrated in the case of M W, a retired businessman of seventy-nine, who developed a coronary occlusion with anterior infarction and did well for two days. On the third day his heart rate suddenly increased to 160. The electrocardiogram showed a supraventricular tachycardia with bundle branch block. The *arrhythmia* was remittent at first but then became constant and the patient developed dyspnea. It was decided, therefore, to administer intravenous Cediland, 4 mg, but, by the time this had been drawn into the syringe, the *arrhythmia* had ceased and did not recur. If the drug had been administered, relief of the *arrhythmia* would have erroneously been attributed to it.

Ventricular tachycardia is a more serious and persistent complication in acute coronary occlusion. It usually responds to Pronestyl or quinidine and these drugs should be given promptly as outlined above (Chapter 1). A par-

ticularly difficult case treated successfully with Pronestyl was M.G., a sixty-year-old tailor with a history of duodenal ulcer who was admitted to the hospital because of melena and hematemesis of several days' duration. For several months he had experienced pain in his left arm on effort and this symptom increased following the onset of bleeding. Physical examination was negative but the electrocardiogram showed an acute coronary occlusion with anterior wall infarction and regular sinus rhythm. His course was uneventful until the twelfth day when he suddenly became dyspneic and perspired profusely. His heart rate was 190 and the rhythm was slightly irregular. The electrocardiogram showed a ventricular tachycardia. The patient received morphine, intravenous quinidine, .65 gm. (10 gr.) on two occasions, intravenous magnesium sulfate and intramuscular quinidine without effect. He developed congestive failure within twenty-four hours and was given Mercurhydri and Digoxin, 1.5 mg. by mouth, with some improvement. He then received quinidine, 4 gm. (6 gr.) every two hours by mouth. The rate of the ventricular tachycardia ranged between 130 and 140. On the third day of the arrhythmia he was given 1 gm. (15 gr.) of procaine amide (Pronestyl) intravenously in fifteen minutes. During the injection a few sinus beats appeared but the tachycardia recurred a short time after the drug had been discontinued. The next day, 2.25 gm. of Pronestyl were injected intravenously in thirty-five minutes and numerous sinus beats appeared. Within two hours regular sinus rhythm appeared and the patient made an uneventful recovery.

Many years ago it was suggested¹⁸⁸ that quinidine be routinely administered early in coronary occlusion to prevent ventricular tachycardia. We do not subscribe to this view because, in our experience with 1000 cases of coronary occlusion who did not receive digitalis, ventricular tachycardia occurred in only .5 per cent.²¹⁰ We, as well as

others, do not believe that it is necessary routinely to employ a drug such as quinidine with potential toxic effects in an attempt to prevent a complication which is infrequent.⁶⁹ We do, however, administer quinidine or Pronestyl if numerous premature beats appear, particularly if they are multifocal.

• Varying degrees of partial or complete A-V block are not uncommon following coronary occlusion.²¹¹ P-R prolongation is usually not significant and remits spontaneously. Partial heart block with dropped beats and complete A-V block occur with diaphragmatic infarction and are more serious. If Stokes-Adams seizures occur, treatment should be carried out as outlined in Chapter 1.

Although Adrenalin is ordinarily avoided in coronary occlusion, it is effective and should be used if Stokes-Adams attacks intervene,⁷ as the following case demonstrates. A forty-nine-year-old man was admitted to the hospital because of recurrent attacks of unconsciousness and substernal pain for two weeks. On examination, he appeared critically ill. His ventricular rate was 28 and the auricular sounds were audible at a rate of 100. His blood pressure was 170/70, as it was being taken, the patient developed convulsive movements and the cardiac sounds became inaudible. He recovered from the attack in a few seconds. An electrocardiogram revealed complete A-V block (Fig 11). He was given Adrenalin (1:1000), 4 cc every two hours. On the third day he developed congestive failure and the electrocardiogram showed flutter fibrillation. He received 6 mg Digtoxin and 2 cc Mercurhydrin intravenously and the Adrenalin was discontinued. The next day he developed another Stokes-Adams seizure, the Adrenalin was resumed every 2 hours. Nevertheless, the attacks recurred several days later and the Adrenalin was given every half hour or hour. He improved and the frequency of the Adrenalin injections was gradually diminished. It was again increased one week later when the attacks recurred, and Adrenalin

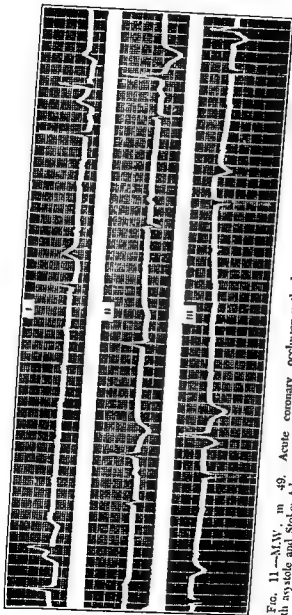


FIG. 11—M.W., m 49. Acute coronary occlusion with diaphragmatic infarction Complete A-V block with asystole and Stokes-Adams seizures Treatment consisted of epinephrine mg 5 to 10 q $\frac{1}{2}$ h for several days

could not be discontinued until two months after admission. Thereafter, the Stokes-Adams attacks did not recur although the complete A-V block persisted.

Anticoagulant Therapy.—Considerable difference of opinion exists today concerning the use of anticoagulant drugs in acute coronary occlusion. After their introduction less than 10 years ago, several reports indicated a significant decrease in embolic phenomena and mortality rate¹⁴¹ and their use became routine in coronary occlusion unless a specific contraindication existed. The rationale for their use was partly based on post-mortem reports which placed the incidence of pulmonary and peripheral emboli in coronary occlusion between 45 and 60 per cent.^{112,114} In the past few years, however, as the result of the clinical observation of increasing numbers of mild cases, it has been suggested that anticoagulant drugs are unnecessary in such attacks.^{214,245} In our experience, the mortality rate in first attacks of coronary occlusion in private patients is less than five per cent. Other observers have found the mortality rate in all mild attacks to be 3.1 per cent and believe that the use of anticoagulant drugs is unjustified in such cases since the incidence of embolic phenomena is very small and accounts for no more than 1 per cent of fatalities.²⁷⁴

Our experience is now in accord with this point of view. In recent years we have not used anticoagulant drugs in mild cases of coronary occlusion and have obtained excellent results.

Anticoagulant drugs are indicated, (1) when congestive failure or shock is present, (2) if there is a history or evidence of peripheral phlebitis, (3) following pulmonary embolism or infarction, (4) following peripheral embolism, (5) in obese or debilitated patients whose movements are restricted or who may require prolonged bed rest. Pulmonary emboli usually originate in the lower extremities and lodge in the lower lobes.²⁰⁵ It is possible that a small percentage arise in the

Table 6.—*Differential Diagnosis of Pulmonary Embolism and Coronary Occlusion.*

	PAIN	DIAPHRAGM	SEVERE SNOKE	HEMOPTYSIS	HEART SOUNDS	PULMONARY RÙ	PULSE	ECG	X-RAY
PULMONARY EMBOLISM	often absent, not typical	often negative	common in cases with large infarct	Not uncommon	P ₂ often accentuated	absent	may be high	a) RS-T depression T-wave inversion, (coronary insufficiency) b) S ₁ , Q ₁ patterns c) Right BBB	Hazy patch in lower lobe may be present
CORONARY OCCLUSION	usually severe, substernal, precordial, left arm	often mild or absent	occurs in 3-10% of cases	absent unless pulmonary edema present	gallop rhythm common	may be present about 10°-15° of rotation	usually not over 101°-102°	RS-T elevation & Q waves Serial changes	may show minimal consolidation

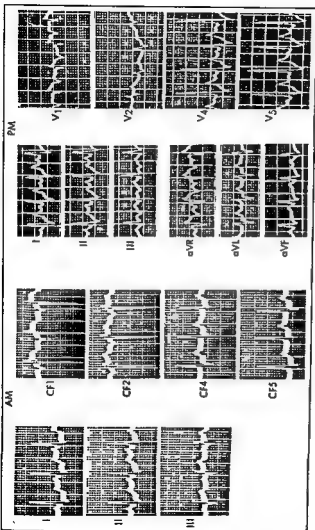


FIG 12—E V, f 40 Acute pulmonary embolism fifteen days following pelvic operation. Death nine hours later. Necropsy revealed bilateral pulmonary embolism of right main and left lower lobe branches. The posterior papillary muscle of the left ventricle showed focal necrosis. AM—Record taken soon after onset showed RS-T depressions. PM—Record several hours later showed prominent S-wave in leads I and II and RS-T rounding in lead III (Dick, Master, Horn, Goldman and Fick, courtesy of Am J Med.)

right auricle in the presence of atrial fibrillation or prolonged congestive failure. The symptoms of pulmonary embolism are very variable. There may be severe chest pain, dyspnea and collapse simulating coronary occlusion, or merely cough, fever or tachycardia (Table 6). Electrocardiographic abnormalities are present in half the cases, the most common single pattern being that of coronary insufficiency, i.e., RS-T depression and T-wave inversion⁶⁷ (Fig 12). Other patterns consist of a deep S₁ and Q₁ with RT₁ elevation, and occasionally right bundle branch block. Pulmonary emboli usually do not occur until after the first week of the attack, most commonly during the second week. About one-third of the patients with pulmonary embolism expire within a few minutes or hours despite all treatment, if a patient survives more than three or four hours he probably will recover.³¹⁸ In some cases it is difficult to distinguish between pulmonary embolization and fresh myocardial infarction. The following points are helpful in making a differential diagnosis. (1) Although fever, tachycardia and chest pain may be present in both conditions, and cough may occasionally occur in cases with coronary occlusion and mild congestive heart failure, the presence of cough and hemoptysis favors the diagnosis of pulmonary embolism. (2) Calf tenderness or a positive Homan's sign¹⁴⁶ indicates the presence of a thrombotic process in a lower extremity and, therefore, possible pulmonary embolization. (3) A loud pulmonic second sound, not previously heard, may indicate pulmonary hypertension secondary to infarction, and the finding of a hazy patch of infiltration on chest x-ray may further suggest the diagnosis of an embolic episode. (4) The electrocardiographic changes described above may be present in pulmonary infarction and simulate a posterior wall infarction but a Q-wave is absent in aVF. All of these findings may help to distinguish between the two conditions. Occasionally, pulmonary infarction occurs during the course of a coronary

occlusion without the appearance of chest pain, cough and electrocardiographic findings. In these instances, a persistent, high fever or leucocytosis may suggest the diagnosis.

If one of the indications for anticoagulant therapy is present, heparin should be given by injection for an immediate effect²⁵⁹ and oral therapy with Dicumarol,¹² Tromexan^{17 136 260} or Hedulin³² begun for a later effect. As soon as the oral medication has prolonged the prothrombin time to therapeutic levels, (in 24 to 48 hours), the heparin is discontinued.

Heparin may be given subcutaneously, 100 mg every eight hours, intramuscularly as Depo heparin, in an initial dose of 400 mg, or intravenously, 50 mg every four hours around the clock. In the above dosage heparin usually prolongs the clotting time to approximately double the control value, i.e., from fifteen to twenty-five minutes (Lee White method) and there is ordinarily no danger of hemorrhage. The clotting time should be determined immediately before, and two hours after, the first dose of heparin, in order to discover patients who are sensitive to it. Thereafter, this determination is unnecessary. If Tromexan or Hedulin is used, it is usually unnecessary to administer more than one or two doses of subcutaneous or intramuscular heparin. If hemorrhage does occur in the course of heparinization, a 1 to 2 per cent solution of protamine sulfate is given by vein in a dose of 1 mg of protamine per mg of heparin.

The administration of oral anticoagulants requires much closer supervision than does heparin, since there is a tremendous variation in the response to them from person to person and, in the same person, from day to day. Even after some stabilization of dosage appears to have been achieved, further administration of the drug in some cases produces unpredictable effects. Therefore, during the first two weeks of treatment the prothrombin time should be determined daily, before the next dose is given. If Dicumarol is used, an initial dose of 300 mg followed by 150 to 200 mg. the

next day is usually quite safe, but we have observed several cases in which one dose of 300 mg of the drug prolonged the prothrombin time to dangerous levels. For example L.W., a woman of twenty-eight with chronic rheumatic valvular disease, mitral stenosis, congestive failure and atrial fibrillation, experienced several episodes of peripheral embolization. Following a recent one, she received 300 mg of Dicumarol on the first day and 200 mg on the second. Her prothrombin time was thirty-six seconds on the third day, and no Dicumarol was given on that day or the next. On the fourth day diffuse ecchymoses appeared over the entire body, and her prothrombin time was ninety-four seconds. She received 2 doses of 72 mg of intravenous vitamin K within five hours and the prothrombin time fell to normal limits within eight hours. An uneventful recovery followed.

M.R., a garage owner of fifty-six, developed a coronary occlusion. He received 300 mg of Dicumarol on the first day and 200 mg on the second. His prothrombin time rose to fifty-two seconds and did not fall during the following two weeks, although he received no more of the drug.

These cases illustrate the extreme variability of response that may occur following Dicumarol therapy.

In Dicumarol therapy, the prothrombin time should be maintained at 2 to 2½ times the control time, i.e., between twenty-five and thirty-five seconds. In most patients this result is achieved with a daily dose of 50 to 150 mg. The urine should be inspected grossly every day and urinalyses should be done every two to three days, if a significant number of red cells are found in the urine, the dosage should be reduced or the drug temporarily stopped. If the prothrombin time is prolonged to sixty seconds or more, or if bleeding occurs, the drug should be stopped and the patient given intravenous vitamin K₁ oxide,²²⁹ in a dose of 50 mg. The emulsion of vitamin K₁ oxide may also be given orally. Usually the prothrombin time will return toward

normal levels within four to eight hours. If K_1 oxide is unavailable, intravenous vitamin K (72 mg) should be given every four hours until the prothrombin time is shortened to safe levels. In severe cases, frequent transfusions of fresh blood are invaluable.

Because of the danger of hemorrhage, anticoagulants should not be administered to patients with a history of hepatic, gastric or renal disease, to those with a hemorrhagic tendency, or in the presence of ulcerative colitis or a cerebrovascular accident.

Occasionally a patient develops hemorrhage while on anticoagulant therapy despite the fact that his prothrombin time has been reported to be within safe limits. A good example of this was the case of a fifty-eight-year-old man who developed a coronary occlusion with anterior wall infarction and was given 300 mg and 200 mg of Dicumarol on the first two days. On the fourth day his prothrombin time was twenty seconds but he passed a large tarry stool, and his hemoglobin fell to 8.6 gm. He was given a small transfusion slowly and recovered quickly.

Hemorrhage may occur into serous cavities, even into the pericardium.^{147,154} It is important to remember that anticoagulants are contraindicated in nonspecific pericarditis since they may cause pericardial hemorrhage. It has even been suggested that they be withheld in coronary occlusion, when a definite pericardial friction rub is present, since pericardial hemorrhage with cardiac tamponade has been observed in one or two such cases following anticoagulant therapy. It is occasionally difficult to differentiate nonspecific pericarditis from coronary occlusion during the first few days of the attack (Table 5); if there is any doubt, anticoagulants should not be administered.

Anticoagulant therapy should be maintained through the period of bed rest and for 5 to 7 days afterward. Some physicians continue it indefinitely for months or years in order to prevent a recurrence of coronary thrombosis.²¹⁷ The value of this procedure has not been proven.

In no instance should oral anticoagulant therapy be instituted unless facilities are available for reliable prothrombin time determinations. Even if these are present, occasional mistakes are made in determining the prothrombin time and, therefore the clinical picture should be closely observed.

Other Oral Anticoagulants.—Of numerous oral preparations recently introduced, only two have proven useful clinically. Tromexan,^{47 116 260} an ethyl ether of Dicumarol, acts much more quickly than the latter drug and is not cumulative. Its effect is apparent in eighteen to twenty-four hours and maximum in thirty hours. Therefore, when it is used with heparin, the latter may be discontinued after the first day. The dose of Tromexan is five or six times that of Dicumarol, *i.e.*, the initial dose is 1500 to 1800 mg. and the maintenance dose 300 to 900 mg. Although its rapid action is desirable, Tromexan has proved to be less satisfactory than Dicumarol, because of the greater variability in individual reaction and in daily maintenance dose and the sudden, unpredictable changes in prothrombin time.^{419 326} In general, therapy with Tromexan requires more careful control than with Dicumarol. *Hedulin* (Phenylindandione)³² also acts more rapidly than does Dicumarol, exerting its maximum effect in twenty-four to thirty-six hours. Its action is dissipated in twenty-four to forty-eight hours. The initial dose is 300 mg. for patients over 150 lbs. and 200 mg. for those weighing less.³² The usual maintenance dose is 50 to 100 mg. The daily dose is divided in two, given in the morning and at bedtime. Final evaluation of the drug is not yet possible because there have been no reports of its use in large, well controlled series of patients with coronary occlusion.

If peripheral arterial embolization occurs, despite anticoagulant therapy, the anticoagulants are continued. In cerebral embolization, however, great caution must be observed in administering these drugs, if a spinal tap does not reveal hemorrhagic fluid, anticoagulant therapy may be

carried on after several days. Sedation should be avoided in these patients and caffeine sodium benzoate, 7½ gr. (5 gm.), intramuscularly every four hours, and aminophyllin 3 gr. (2 gm.), intramuscularly every four hours, may be given. Other supportive measures should be employed, oxygen should be used, and parenteral feedings continued until the patient is able to feed himself.

When a peripheral embolism occurs, intra-arterial Priscoline, 50 to 75 mg., every four to six hours, given proximal to the occlusion, is sometimes of value in increasing circulation in the affected extremity.²⁰⁰ Various ganglionic blocking agents also produce a high degree of vasodilatation and reduce vasoconstrictor tone when administered subcutaneously or intravenously. We have found that Hexamethonium (Bis-trimethyl-ammonium hexane dibromide), produces a longer lasting blockade than other presently available drugs.²⁰¹ Hexamethonium (25 mg./cc.) may be administered in doses up to 1 to 2½ cc. subcutaneously or intravenously every four to six hours to maintain an adequate blockade. The drug should be given cautiously in patients with hypertension. It is best to begin with small doses in order to avoid a precipitate drop in blood pressure. The patient should remain recumbent one and one-half to two hours following each injection. In our experience this type of therapy has largely replaced nerve blocks in these patients since the performance of the latter in patients on anticoagulant therapy may induce hemorrhage. If the patient is able to tolerate oral therapy, the measures outlined above may be supplemented by the use of Priscoline, 50 to 75 mg., or Dibenzyline, 10 to 20 mg., every four hours.^{129, 234} If the patient is seen after gangrene has occurred, the leg should be packed in ice, in preparation for amputation.

Embolectomy is the treatment of choice if it can be performed within eight to twelve hours after the acute episode. In expert hands results have been good, and even

massive saddle emboli lodged at the bifurcation of the aorta have been removed successfully¹⁶⁴ It is a serious operative procedure, particularly in patients with recent myocardial infarction, but it is justified unless the patient is critically ill A patient recently observed illustrates the fact that embolectomy can be performed successfully, even in the presence of serious heart disease

A sixty-three-year-old tailor developed an acute coronary occlusion with anterior infarction He did not receive Dicumarol On the fifth day he complained of severe pain in the right leg which became mottled and cold up to the mid thigh The femoral pulse was not palpable Five hours after the onset of symptoms operation was performed by Dr. Gabriel Seley, of New York and an embolus was removed from the femoral artery Complete function of the extremity was restored, and the patient made an uneventful recovery

If embolectomy is not feasible and amputation becomes necessary, it should be performed under local or block anesthesia, after demarcation has occurred

Length of Bed Rest—There has been considerable difference of opinion as to how long patients with acute coronary occlusion should be kept at strict bed rest. For many years a period of six weeks in bed was considered essential in all cases in order to allow a "firm" scar to form²⁰³ and at present many physicians insist upon four weeks lest a ventricular aneurysm develop²⁰⁴

Recent observers however, have emphasized the fact that prolonged bed rest is often unnecessary in acute coronary occlusion or heart failure, and, in fact may have harmful effects.^{76, 125, 184, 197, 200} It has been shown that recumbency tends to increase cardiac work by raising the circulating blood volume.²⁵⁸ In addition to producing serious psychological tensions, prolonged bed rest causes generalized loss of muscular and vascular tone, constipation and distention, and predisposes to venous thrombosis⁷³

For these reasons many physicians now allow patients running a mild course to use a commode early in the attack and to sit in a chair after one or two weeks. Others however, have advocated the armchair treatment from the first day or two, particularly when *heart failure* is present but not when severe, prolonged shock or cerebral or peripheral embolism exists^{184,185}. Follow-up reports indicate that patients so treated do not develop ventricular aneurysms more frequently than do others²³⁰. It should be emphasized that the armchair regimen does not involve early ambulation, the patient is helped or lifted into a chair and ■ returned to bed in the same way after a number of hours. This treatment of acute coronary occlusion will have to be evaluated over a longer period with well controlled studies before a final opinion may be reached.

At the present time each patient should be treated *individually*. It is evident that the mild case may be treated liberally and may be expected to make a good recovery. If such a patient objects to the bedpan, he may be permitted to use a bedside commode and, once he is up, to sit in a chair. On the other hand, if a patient running a mild course does not mind a short stay in bed, we are inclined to keep him in bed one week and then to permit him to sit in a chair. While in bed, he is advised to move his legs and may feed himself.

A more important decision must be made, in respect to the armchair treatment, in patients who are more seriously ill with congestive failure, tachycardia, persistent hypotension of 80 or less or a state of mild shock. Such patients should be treated in bed with the usual therapeutic measures. If failure ■ present, every effort ■ made to approximate the sitting position by using a hospital type of bed or by placing 9 inch blocks under the headposts. If the patient fails to improve, the effect of placing him in a chair may be determined.

Ambulation—If the course of a coronary occlusion has been mild and the patient has been sitting in a chair during the first week, we usually permit him to walk by the third week. Such a patient is usually ready to return to work in two or three months. In the more severe cases a patient may not be ready to walk for four to six weeks or to resume working for three to six months. Four out of five patients who have recovered from a coronary occlusion lead productive lives for many years.²¹⁹

We do not depend upon the electrocardiogram or sedimentation rate to determine the severity of the attack or when the patient may sit up, walk about or go back to work. If the patient is asymptomatic and the clinical findings are satisfactory, ambulation is permitted although the electrocardiogram shows marked alterations. In some cases, we do not wait for the electrocardiogram to become stable. While a very abnormal sedimentation rate is of diagnostic value early in the attack, we have observed serious cases with little elevation, as well as cases with a high sedimentation rate for months or even years during which time they have been active and well.

A normal ballistocardiogram following coronary occlusion usually indicates a good recovery but the patient may be doing very well when his ballistocardiogram is definitely abnormal.²²⁷ It must be remembered that the ballistocardiogram may be abnormal in people over 50 without heart disease.

Nausea, Vomiting, Distension and Hiccough—These are common disturbing and serious symptoms in acute coronary occlusion. The suggestions for prevention and treatment outlined in Chapter 2 should be followed.

Chapter

4

SYNCOPE

SYNCOPE or fainting is a common symptom and occurs in many non-cardiac conditions (Table 7). Only occasionally is it caused by organic heart disease or cerebral arteriosclerosis. Syncope is the result of temporary cerebral anoxia which is most frequently produced by a marked decrease in peripheral resistance and fall in blood pressure, the cardiac output being fairly well maintained. This type of syncope or fainting is termed neurogenic or vasodepressor.¹⁰² Fainting occurs because in the upright position pooling of blood occurs primarily in the lower extremities. A less frequent mechanism of cerebral anoxia and syncope is a sudden drop in cardiac output, as occurs following severe hemorrhage.⁴² Not infrequently both mechanisms, *i.e.*, a drop in blood pressure and in cardiac output, are present. Other specific causes of syncope include postural hypotension, hysteria and anxiety states, hypoglycemia and severe coughing bouts²⁷¹ or bearing down efforts.

It is usually possible to determine the type of syncope if the characteristics of each are borne in mind (Table 7).

(a) *Neurogenic or Vasodepressor Syncope*—This is the commonest type of syncope. It is associated with reflex peripheral vasodilatation which may be precipitated by the sight of blood, fright, pain or a severe emotional disturbance.²⁸ Not uncommonly it is induced in the doctor's office by the sight of a hypodermic needle for an injection or at the Red Cross or a hospital when the needle is to be inserted

Table 7.—Differential Diagnosis and Treatment of Syncope

Type	Precipitating Factor	Prodroment Symptoms	Duration of Attack	Faintness	Treatment
1) Vasodepressor Syncope a) Neurogenic	Friction, pain, emotional upset, sight of blood	Nausea, sweating, light-headedness, dizziness, body occurs when patient stands, presyncope phase 1-5 minutes	Usually of short duration, 1-15 seconds after 15 seconds twitching may appear	Cardiac output normal Blood pressure falls Pulse rate may rise or fall Electroencephalogram abnormal	Usually terminated by laying patient down with head lower than feet Rarely .5 to 1 cc Epinephrine (subcutaneously) if necessary
b) Carotid Sinus Syncope	a) Pressure on neck b) Turning head c) Circular or circumflex rotation d) Distal tie	Usually no prodroment symptoms	Short duration Convulsions may occur	Blood pressure falls Pulse rate decreases Electroencephalogram abnormal	Epinephrine Sulfate 25 mg t.i.d. Probolazine 15 mg or Atropine 3 mg t.i.d. Injection of sinus nerves if attacks are severe and persist
c) Secondary to Other Factors	Internal or external hemorrhage, α or β to peptide toxin	Same as (a)	Same as (a)	Cardiac output diminished	Treat primary condition
2) Postural Hypotension	a) Tablets dorsalis b) Spinal cord tumors c) Adhesive discus d) Distal tie inclining e) Standing in one place, arising suddenly f) Idiopathic	Light-headedness, dizziness	Usually of short duration (1-5 seconds) No convulsions	Blood pressure falls Pulse rate increases Electroencephalogram abnormal	lay the patient down Elastic stockings, light alkaline diet and xeromynephrine or epinephrine Tilt bed, high Na diet, DOCA (Caution against raising quickly) Treat primary disease
3) Hypertensive Syncope	a) Suggestion b) Pressure on any area of body c) Tense situation d) Hyperventilation	No prodroment symptoms	Never any convulsions May last for hours	No change in blood pressure or pulse rate No electroencephalogram changes	Psychiatrist
4) Cardiac Syncope	a) Aortic stenosis b) Heart block with periods of systolic collapse c) Ventricular tachycardia	Dizziness, palpitation, chest pain, nausea, epigastric pain	Usually about 15-10 seconds May last longer with convulsions	Electrocardiographic abnormalities Blood pressure falls Pulse rate may be extremely slow rapid or uninterpretable	For cardiac arrhythmias, Quinidine, Digitalis, Prostig For heart block, Epinephrine, Isuprel, Atropine, Pacemaker
5) Hypoglycemic Syncope	a) Islet cell tumor b) Obstructive pancreas c) Too great an interval between meals d) Hepatic disease	Weakness, extreme hunger, tremors, sweating, pallor	May be preceded if not treated Convulsions occur frequently	Blood sugar extremely low Electroencephalogram abnormal Pulse rate increased Blood pressure unchanged or low	Intravenous glucose, then oral glucose—depending upon etiology (see text) a) remove tumor if present b) low carbohydrate diet c) high carbohydrate diet if liver disease is present
6) Toxic Syncope	a) Severe coughing b) Hoarse coughing	Dizziness, lightness	Short (2-5 seconds)	No unusual findings	Warn patient against coughing, or excessive coughing while standing

in the arm during blood donation. Syncope resulting from peripheral vasodilatation may also be observed in people who, while sitting or recumbent, employ *heating pads* for low back pain, or who have been sitting or reclining in the hot sun. When they suddenly assume the erect position, temporary cerebral anoxia occurs and fainting may result. Syncope also occasionally occurs in normal persons who are required to stand in one place for long periods of time.

Vasodepressor syncope is more frequent in men than in women. Premonitory symptoms such as nausea, sweating, or lightheadedness usually precede the attack, which occurs only if the patient is standing. This type of syncope can usually be terminated by having the patient lie down with his head lowered. This is far more effective than placing the head between the knees. If the syncope lasts more than ten to fifteen seconds, clonic twitching may appear, but true grand mal seizures are extremely rare. In this type of attack it is sometimes necessary to administer vasoconstricting drugs, e.g., epinephrine, 5 cc of a 1:1000 solution, subcutaneously, or neosynephrin, 5 to 10 mg, intramuscularly.

An important type of vasodepressor syncope to bear in mind is encountered in persons with a hypersensitive carotid sinus. It is not uncommon for such people to faint when the sinus is massaged²⁹⁴ but only a small percentage do so spontaneously. Consequently, a diagnosis of carotid sinus syncope should be made only if the following criteria are satisfied: (1) The attacks must occur without the premonitory signs usually seen in other forms of vasodepressor syncope, e.g., those associated with fright or the sight of blood. (2) The attacks should be reproducible by sinus stimulation. (3) The attacks should not occur after the administration of adequate doses of atropine or ephedrine, i.e., doses large enough to prevent bradycardia on manual carotid sinus stimulation. True cases of carotid sinus syncope are relatively uncommon when compared with other types of vasodepressor syncope.

An interesting illustration of carotid sinus sensitivity was furnished by a seventy-one-year-old man who was admitted to the hospital because of four attacks of syncope during the preceding four months. Examination revealed a blowing

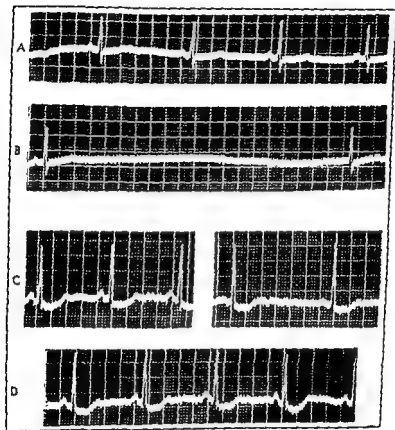


FIG 13—JQ, m 61 Arteriosclerotic heart disease Continuous lead II A, Control, sinus bradycardia, rate 52. B, After pressure on left carotid sinus Asystole for four seconds followed by nodal beat C, Following oral atropinization and compression of right carotid sinus D, Compression of right carotid sinus following intravenous atropinization

systolic murmur at the aortic area, transmitted into the neck. The blood pressure was 160/80. The peripheral pulses were not palpable. The electrocardiogram showed evidence of a previous lateral wall infarction. In the hospital he experienced brief episodes of dizziness. At first it was thought that his syncope was the result of cerebral arteriosclerosis and aortic stenosis. Pressure on the right carotid sinus, however, produced asystole and a drop in blood pressure to 80/60. The patient became blanched, his hands twitched and he yawned. Stimulation of the left carotid sinus produced similar results, but with less regularity. He was given oral atropine, 3 mg. (gr 1/100) q i d. and, after several days, carotid sinus pressure failed to cause a change in his heart rate (Fig 13). The patient has been maintained satisfactorily on this medication. It was difficult to establish a diagnosis of true carotid sinus syncope in this case, despite the fact that definite hypersensitivity of the sinus could be demonstrated, since the patient had cerebral arteriosclerosis and aortic stenosis, which alone may have caused the syncope. Since atropine definitely prevented the attacks, however, the diagnosis of syncope secondary to carotid sinus sensitivity was the most logical one.

Patients who have extensive arteriosclerosis or are receiving digitalis are more susceptible to syncopal attacks and, in some instances, stopping digitalis eliminates the attacks completely. Carotid sinus syncope may be induced by pressure on the sinus from any cause, e.g., by an enlarged gland in the neck, by a sudden movement of the head while shaving, and by sitting with one's head leaning on his hand.

Tight collars may serve as a trigger mechanism. J. C., a thirty-eight-year-old midwestern preacher, was investigated because he had fainted several times while delivering his Sunday sermon. In eliciting his history it was discovered that during the week he worked in the field with his collar open,

on Sunday, however, he wore a stiff collar to appear in church. Carotid sinus pressure repeatedly induced asystole and syncope in this patient, and the attacks were eliminated when the patient stopped wearing tight collars.

Convulsions may occur as a result of excessive carotid sinus stimulation¹⁴¹ and caution must be observed when testing a patient for sensitivity, particularly an older person with arteriosclerosis. Pressure should be applied unilaterally for not more than ten seconds and the patient should be recumbent when tested. Ephedrine sulphate (25 mg.), Paredrine (60 mg.) or atropine sulphate three times a day prevents most attacks. Atropine is given in tablets of gr. 1/50 to 1/100 or 10-15 gtt Tr. Belladonna until dryness occurs. Recently Banthine (50 mg.) or Pro-banthine (15 mg.) three times a day has been advocated. Neosynephrine, in oral doses of 10 to 20 mg. three times a day, is occasionally successful when other medications fail. When it is necessary to use sympathomimetic drugs, i.e., ephedrine, for long periods of time, it is wise to administer small doses of a sedative (barbiturates) simultaneously to prevent nervousness and insomnia. In a few cases, excision of the sinus has given permanent relief.¹⁴³

(b) Postural hypotension is associated with poor sympathetic nervous regulation of blood vessels.^{40,303} It may occur in normal persons, in patients with neurocirculatory asthenia and in those with serious underlying central nervous system disease,³⁴ such as cord tumor or tabes dorsalis.²⁹⁸ It is common following sympathectomy and the use of autonomic blocking drugs for the treatment of hypertension. We have also observed extremely severe postural hypotension in patients with advanced diabetes mellitus, Addison's disease and pheochromocytoma. When these people assume the upright position, blood is pooled in the lower extremities and, as a result, the normal compensatory splanchnic constriction does not occur, the cardiac output is diminished and the blood pressure falls. Hyperventilation

has recently been suggested as a factor in postural hypotension syncope⁴⁸

In patients with chronic postural hypotension and recurrent syncopal episodes, therapy is extremely difficult. A primary disease should be sought and treated if possible, *i e*, Addison's disease. If no underlying condition is found, the most effective method of prophylaxis is the application of elastic bandages around the legs and thighs. A tight abdominal belt may also be helpful. Atropine, epinephrine, neosynephrin or Paredrine may be tried, in the doses cited, but have not proven very successful. They are usually more effective when used in conjunction with leg or abdominal binders. At times it may be temporarily helpful for these patients to sleep in a bed tilted 20 to 25 degrees from the horizontal with the head up,²⁹¹ in order to train the vasomotor system to vasoconstrictor activity. Desoxycorticosterone acetate and sufficient salt to cause slight dependent edema sometimes reduce the frequency of attacks. The patients should be instructed to rise from bed slowly and to avoid taking drugs that cause peripheral vasodilation, *i e*, amyl nitrite or nitroglycerin.

A severe instance of postural hypotension was recently observed in a forty-two-year-old woman. She also exhibited anhidrosis and miosis which suggested a central type of sympathetic nervous system defect, presumably in the hypothalamus. An identical clinical picture has been noted by us in patients receiving chemical sympathetic blocking agents, *e g*, Dibenzyline. A response to sympathomimetic drugs was not obtained, however, in this patient, and it was necessary to maintain her on six to eight grams of added salt and 5 to 10 mg of Desoxycorticosterone daily. She has done remarkably well on this regimen although ankle edema has appeared intermittently.

(c) Fainting may be *hysterical* in origin, particularly in women. This type of syncope is not accompanied by premonitory symptoms and is not associated with any.

changes in pulse or blood pressure. These patients may fall to the ground and remain unconscious for a period of several seconds or hours, but convulsions do not occur and injury rarely results. Electroencephalographic changes, which are seen in most other forms of syncope that last more than a few seconds, do not appear in this condition.²⁶ Fainting may be precipitated by suggestion or by manipulation of almost any part of the body. Therapy is mainly psychiatric.

In anxiety states syncope occasionally occurs as a result of *hyperventilation*. Such an attack may be reproduced by having the patient breathe deeply for one or two minutes. Assurance and sedation are the only treatment of value in correcting this condition.

(d) A rare form of syncope is that which follows severe bouts of *coughing* or the Valsalva maneuver (a forced expiration with the glottis closed—the “bearing down” effort).^{2,20} A marked rise in intrathoracic pressure occurs and prevents adequate filling of the right atrium, resulting in a marked fall in cardiac output and cerebral anoxia. Recognition of this type of syncope is important since most of these attacks can be prevented by warning the patient against excessive bearing down efforts, such as straining at stool, and against remaining upright during a spell of severe coughing.

(e) Fainting which occurs early in the morning before breakfast or several hours after a meal may be the result of *hypoglycemia*. This type of syncope is seen in cases of hyperinsulinism caused by pancreatic adenomata, in patients on a high carbohydrate diet with an overactive pancreas, and in cases of advanced hepatic disease.²⁸ The patient usually experiences weakness, dizziness and extreme hunger before fainting, convulsions often occur if therapy is not instituted quickly. Intravenous or oral glucose should be given as soon as possible and recovery from the attack is usually rapid. The diagnosis is made from the history, the fasting blood sugar determination and the glucose tol-

erance test. Extremely low blood sugar levels may be obtained during the course of the latter test, particularly in the two-, three-, and four-hour specimens, and fainting may occur before the test can be completed. These attacks may be prevented in several ways, depending upon the etiology of the condition. (1) If hypoglycemic attacks occur before breakfast, and rarely during the day, and if liver damage is demonstrated, a high carbohydrate, high protein diet should be prescribed. (2) If the attacks occur before breakfast and frequently during the day, and if they become progressively more severe and are not relieved when the patient is put on a low carbohydrate, high protein diet, a pancreatic adenoma is probably present and surgical removal of the tumor is advisable. (3) If the attacks occur approximately two to three hours after meals, and if no evidence of liver disease or progression is found, so-called "functional" hypoglycemia is present, and the patient should be put on a low carbohydrate, high protein diet with frequent feedings. Additional points of differentiation between these three major types of hypoglycemia and suggestions for therapy may be found in a recent review.⁵⁵

(f) In *organic heart disease*, syncope may occur in complete A-V block or calcific aortic stenosis (Chapter 1) and, rarely, it may usher in an acute coronary occlusion. It is not uncommon at the onset of any type of paroxysmal tachycardia, whether functional or associated with heart disease.⁵⁷ For example, a man of forty-nine was recently hospitalized for recurrent syncope of unknown etiology. After several days he fainted and, when he was seen, almost immediately, atrial fibrillation was present. It ceased in a few minutes. The prognosis of syncope naturally is more serious in heart disease than in non-cardiac conditions.

Cardiac syncope is most frequent in *calcific aortic stenosis*, whether rheumatic, congenital or sclerotic, and usually follows exertion. It should always be kept in mind as a possible cause of syncope. A pertinent case was a woman

who began to experience occasional syncope in her eighties. A harsh aortic systolic murmur was present but her hemoglobin was less than 10 gm., and it was thought that the anemia was the cause of her syncope. However, although the anemia responded to therapy, she continued to experience syncopal attacks and the aortic murmur increased in intensity. After several years she developed recurring Stokes-Adams seizures and died within several days, doubtless as a result of the aortic stenosis.

In the past, aortic stenosis resisted all treatment once it had advanced beyond a certain stage. Recently, however, aortic valvulotomy has been performed in several hundred patients.⁹ Although satisfactory results have been reported by some authors, the mortality rate of this procedure is quite high and it requires further evaluation.

(g) *Cerebrovascular Disease* Vertigo and syncope are not infrequent in the course of chronic cerebrovascular disease, and at times may indicate the occurrence of a small hemorrhage or thrombosis. Such patients should be observed and treated symptomatically.

Chapter

5

RHEUMATIC FEVER AND HEART DISEASE

DURING the acute stage of rheumatic fever the occurrence of acute pericarditis, congestive failure and, occasionally, arrhythmias necessitates emergency treatment. In such cases early adequate treatment often reduces the morbidity and mortality rate. Previously, only salicylates and symptomatic treatment were available in rheumatic fever. With the advent of ACTH and Cortisone, it seemed at first that a dramatically effective form of therapy had been found³⁴⁶. At present, however, medical opinion is fairly well distributed among those who claim that these drugs are very effective, those who claim that they have little or no advantage over salicylates and those who believe that they may be effective but that this has not been proven as yet³⁴⁷. It is generally agreed that these hormonal drugs suppress the inflammatory reaction in rheumatic fever. Fever and arthritis usually subside in one to three days and the sedimentation rate returns to normal in 10 to 20 days. Tachycardia and gallop rhythm are less consistently affected. Some authors have claimed that the adrenal cortical hormones also suppress severe carditis if administered early, but the majority have not found this to be true. Murmurs have been observed to develop in patients treated with these drugs³⁴⁸. Although the question is not yet settled, it appears that ACTH and Cortisone do not prevent the development of chronic valvular disease following rheumatic fever.

While their action in rheumatic fever remains a moot question, ACTH and Cortisone should be used when the salicylates appear to be exerting little effect or when the symptoms are severe. They should be continued for at least two to three weeks. The initial daily dose of ACTH is 100 mg, of Cortisone, 200 mg, and of Hydrocortisone, 100 mg. If these drugs are effective after several days, the dose is gradually reduced to a maintenance dose, which is usually one-fourth to one-half the initial dose. Since they cause sodium retention and potassium loss, the patient should be placed on a low sodium diet and fruit juices given liberally for their potassium content. Potassium also may be given by mouth.

Congestive Failure.—Although the superiority of ACTH and Cortisone over salicylates has not been proven in the ordinary case of rheumatic fever, they are very valuable in a number of patients severely ill with congestive failure,^{279,344} as the following case demonstrates.

A man of 29 with rheumatic pericarditis was in severe left and right heart failure which did not respond at all to the usual therapy. Marked tachycardia and gallop rhythm were present. After receiving 25 mg of ACTH every six hours for six days, he improved remarkably. The daily dose of ACTH was then reduced from 100 to 60 mg, and this was maintained for seven days. It was then further reduced to 40 mg. This treatment was continued for two more weeks and the patient made an excellent recovery. In this patient, heart failure was relieved only when the adrenal cortical hormones were used. It was our opinion that without them death would have occurred.

The beneficial effect of ACTH and Cortisone in acute rheumatic fever complicated by congestive failure is particularly important since digitalis is often ineffective in this situation³²³ and may even be harmful since there is a tendency to push the dosage of digitalis to toxic levels in order to obtain an effect.²⁶³ It is, therefore, wiser to depend

upon salt restriction, mercurial diuretics and hormonal therapy in congestive failure before digitalization is attempted. If the failure persists, digitalis should be administered for it may be effective, particularly if atrial fibrillation is present.^{32d} Its administration should be controlled with particular care to avoid intoxication.

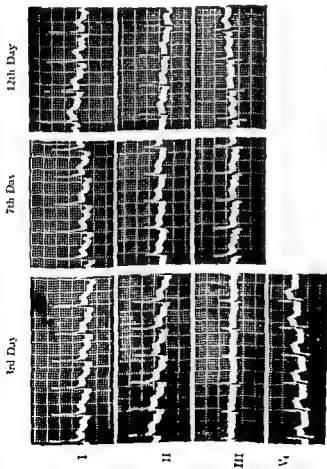


Fig. 14—NIP f 26. Acute rheumatic fever with pericarditis. ST-T elevations are present in all leads.

In the usual attack of rheumatic fever, *salicylates* should be given in large daily doses of 3 to 6 gm. This amount is given in divided doses every four hours. Salicylates are quite effective in controlling the fever and joint pains in acute rheumatic fever, and may have a favorable effect upon the exudative phase of the disease, *i e*, pericardial and joint effusions.¹⁴⁸

Acute Pericarditis.—Acute pericarditis is common in acute rheumatic fever and may be associated with severe precordial or epigastric pain which is relieved only by morphine. It usually develops within a few days after the onset of the attack. A friction rub may be heard and, rarely, shock may occur. The electrocardiogram usually presents a typical pattern consisting of RS-T elevation in two or more leads, without Q-waves (Fig 14) and often with subsequent development of T-wave inversion. If an effusion accumulates, the rub may disappear and the patient usually becomes more dyspneic and orthopneic. Should these symptoms increase and the venous pressure rise, the picture of congestive failure is simulated. Tapping of the pericardium is rarely necessary, however, and is required only if cardiac tamponade develops rapidly. Usually the fluid is resorbed spontaneously as the acute infection subsides on bed rest and drug therapy. In these cases, as in congestive failure, ACTH and Cortisone may be more effective than the salicylates and should be used.

In the course of *chronic rheumatic heart disease* with mitral stenosis and atrial fibrillation, atrial thrombi may form and, in rare instances, may be large enough to occlude the valve opening (ball valve thrombus).^{83,248} This produces a real emergency since blood is unable to enter the left ventricle and the cardiac output is greatly diminished. Cyanosis appears at the extremities of the circulation, *i e*, the tip of the nose, the lobes of the ears and the fingers and toes, and severe dyspnea, congestive heart failure and shock usually develop. In rare instances the thrombus may

be demonstrated by angiocardiology, a radiolucent area being noted in the atrial chamber. Death may occur suddenly⁶ or the patient may live for many years. The treatment is supportive including oxygen and the usual measures employed in congestive failure and shock. Morphine, 15 mg. ($\frac{1}{2}$ gr.), may relieve the restlessness. Occasionally, a change in position causes the thrombus to move and relieves the condition. We have observed the cyanosis to disappear immediately when the patient sat up and leaned forward. Surgical removal of the thrombus presents technical difficulties but seems feasible if the newer methods developed in cardiac surgery are employed, i.e., cross circulation, artificial heart or hypothermia.

Thrombi in the right auricle¹⁴³ may be carried to the lungs, causing pulmonary infarction, and those from the left auricle may be released into the blood stream, resulting in cerebral or peripheral arterial embolization. Emboli occur more commonly in isolated mitral stenosis than when it is combined with mitral insufficiency. The treatment of these vascular accidents has been discussed in Chapter 3. In peripheral embolization embolectomy is the procedure of choice if the diagnosis is made within several hours.

In rheumatic heart disease one embolic episode is apt to be followed by others. Therefore, an attempt should be made to prevent a recurrence. This may be done either medically or surgically. Medical treatment consists of administering an anticoagulant such as Dicumarol over a number of years.^{65, 217} We have observed such a patient over three years during which time not a single embolic episode has occurred, whereas she had developed many of these prior to the institution of Dicumarol therapy. The patient takes 50 mg. of the drug five days a week and her prothrombin time is determined once a month. It is easily controlled in this patient, but others require much closer observation. The surgical procedure originally performed for recurrent peripheral embolization was left auricular appen-

dectomy,^{31 71 203} but recent studies have demonstrated that thrombi form in the atrial cavities as well as in the appendages and that unilateral resection may not be effective.^{163,322} The procedure is reserved for cases in which contraindications to valvulotomy exist.²³⁹

In recent years the development of mitral valvulotomy or commissurotomy as a safe and effective operative procedure has revolutionized the management of chronic rheumatic valvular disease with mitral stenosis.¹⁷⁴ Many patients with even marked restriction of activity and signs of congestive failure have been restored to relatively good health following operation and are able to work. The mortality rate is now less than 15 per cent.

While there is still no unanimity of opinion as to all the indications for mitral valvulotomy and left auricular resection, the operation should be considered in every patient with mitral stenosis. The following indications are generally accepted (1) symptoms of cardiac insufficiency, *e g*, increasing dyspnea and fatigue or *attacks of pulmonary edema*, (2) signs of congestive failure, whether mild or severe, (3) auscultatory, roentgenologic, electrocardiographic and vectorecardiographic evidence of mitral stenosis and right ventricular enlargement, (4) increased pressure in the pulmonary artery and right ventricle on catheterization, (5) *recurrent embolic episodes* and (6) *recurrent hemoptysis*.

Some physicians perform commissurotomy in all patients with definite mitral stenosis before the advent of symptoms of heart failure and of increased pulmonary artery pressure. It must be remembered, however, that many patients with mitral stenosis remain asymptomatic for many years and live out their normal life span. Although it is not possible to predict which patients will run such a benign course, it would seem advisable to perform the operation only when there is a specific indication until the results of the procedure have been observed over a longer period of years.

On the other hand, it is important not to delay the operation until the patient is quite ill, since the results are better in patients with mild degrees of failure than in those in advanced failure.

The major contraindications to mitral valvulotomy are the presence of acute rheumatic fever or subacute bacterial endocarditis and significant left ventricular enlargement, whether caused by mitral insufficiency or aortic valve disease. A mild degree of mitral insufficiency or a minimal aortic lesion does not preclude the operation, but the finding of definite left ventricular enlargement on fluoroscopy or of left ventricular hypertrophy in the electrocardiogram makes the case unacceptable. One of the major difficulties in this field is the detection of mitral insufficiency in patients with mitral stenosis. Experience has shown that the presence of an apical systolic murmur is not in itself a reliable sign of mitral insufficiency, unless it is at least of grade 3 intensity.¹⁵⁶ At operation it has been found that pure mitral stenosis not infrequently exists without insufficiency. Yet in one such series¹⁵⁷ a systolic murmur of grade 1 or 2 intensity (rarely 3) was present in 30 per cent of these cases. A grade 4 apical systolic murmur indicates definite mitral insufficiency, and in this lesion the murmur usually occupies all of systole and may include the second sound. A number of complicated procedures have been suggested for differentiating mitral stenosis and insufficiency but they have not proven superior to auscultation, fluoroscopy and electrocardiography.²

Atrial fibrillation and advanced age (over 50) are not contraindications to mitral valvulotomy.

While a history of arterial embolic phenomena is sometimes considered a contraindication to mitral commissurotomy, because of the probability of postoperative embolism, our experience and that of others in such cases has been favorable. For example, three years ago a woman of 48 with long-standing mitral stenosis and frequent episodes

of paroxysmal atrial fibrillation developed a popliteal embolism. The embolus was removed surgically within three hours with complete restoration of circulation. Six weeks later mitral valvulotomy was performed. Post-operatively she developed atrial fibrillation which persisted for 12 days. Since then she has been asymptomatic except for intermittent premature beats and occasional brief episodes of atrial fibrillation which are readily controlled with quinidine.

We, therefore, advise commissurotomy in patients with a history of embolic episodes. Embolization to the brain during the operation may be prevented by momentarily occluding the left common carotid and the innominate arteries by pressure at the instant of valve fracture.⁴⁷

Mitral valvulotomy has a low morbidity and mortality in the hands of an experienced team consisting of surgeon, cardiologist and anesthesiologist working in close cooperation during and after the operation. During the operation it is advisable to follow the rhythm of the heart electrocardiographically in order to detect arrhythmias or cardiac arrest (See Chapter 9). Postoperatively, severe pain in the area of the incision, arrhythmias and peripheral embolism may occur. Precordial pain, dyspnea, tachycardia and fever ("post-commissurotomy syndrome") have been noted during the first three to six weeks after the operation, but they may be delayed and may constitute an emergency.⁴⁸ It is not clear whether the attack represents rheumatic reactivation or a pleuro-pericarditis, but the symptoms usually subside after four or five days without specific therapy.

Recently surgical procedures for the correction of aortic stenosis and of aortic and mitral insufficiency have been performed. Favorable results have been reported by several groups, but it is still too early for definite evaluation of these operations.^{9, 161} Furthermore, double lesions, either of the same valve or of both the mitral and aortic valve, have been corrected at one sitting in some patients. However, such a procedure should be undertaken only if it is certain

that both lesions are causing a significant circulatory disturbance. For example, when both aortic and mitral stenosis are present, it may be necessary only to correct the mitral lesion to achieve clinical improvement. The introduction of the techniques of hypothermia, cross-circulation, and the artificial heart in cardiac surgery has opened untold possibilities in this field, and continued advances in correcting valvular defects in rheumatic heart diseases are inevitable.

Chapter

6

HYPERTENSIVE ENCEPHALOPATHY AND CRISES—PHEOCHROMOCYTOMA

HYPERTENSIVE crises may occur in the course of essential hypertension, particularly in its malignant phase, in acute nephritis and in cases of chronic nephritis with uremia. Severe unremitting headache, nausea and vomiting, paraesthesias, convulsions, blindness, stupor or coma may be observed. In some instances these episodes are terminal and regardless of therapy the patient will not survive. Relief of symptoms is the most that can be expected from treatment. In other instances the crisis is temporary and represents an exacerbation of a disease which may be in its early stages or which may be reversible. This latter group of patients should be treated as quickly and vigorously as possible, for recovery may be followed by many months or years of comfortable living.²³²

The symptoms which occur are most probably secondary to increasing blood pressure and cerebral edema. Relief from these attacks has occasionally been obtained by the intravenous administration of 20 to 40 cc. of a 10 per cent solution (2 to 4 gms.) of magnesium sulfate, or 50 cc of 50 per cent dextrose or by the withdrawal of spinal fluid. Results with these dehydrating procedures do not compare, however, with the dramatic response noted in some patients when sympathetic blocking or other "anti-hypertensive" agents are employed. Many of these drugs

have been used to relieve the symptoms of hypertensive encephalopathy and prevent permanent cerebral, renal or ocular damage

Although use of the parenteral Veratrum derivatives (Veriloid and Protoveratrine) may occasionally produce good results,^{223 246} most of our own experience has been with the Dibenamine derivatives, Hydrazinophthalazine, (Apresoline) and Hexa- and Pentamethonium.²²³ Intravenous Dibenzyline (5 to 10 mg /kg given in an infusion of glucose in water over a twenty-to-thirty-minute period) will often produce dramatic relief of headaches and a fall in blood pressure. Mental symptoms may be relieved for several days after one dose of this drug ²²³ We do not believe, however, that it is the drug of choice since a definite effect is not always obtained, and if a severe, prolonged hypotensive reaction does occur, none of the presently available pressor agents are effective in counteracting the hypotension. Apresoline (20-50 mg intravenously) will also produce a fall in blood pressure in patients with hypertensive encephalopathy but it may aggravate the headache, nausea and vomiting

At the present time we believe that the use of parenteral Hexamethonium (Bisrium) is the most satisfactory method of treating these patients during the acute episode. This drug must be given slowly and the dose necessary to produce an effect must be determined in each patient. In general, patients with the highest pressures prove to be the most sensitive to drug action. It is best to administer the first dose intravenously as an infusion (100 mg of Bisrium in 100 cc of glucose in water given at a rate of approximately 1 cc /minute). Blood pressures should be checked every minute with the patient in a sitting position. As soon as a blood pressure fall to the desired level $\frac{(150 \text{ to } 170)}{(100 \text{ to } 110)}$ occurs, the infusion should be stopped and the patient lowered to a recumbent position. If this method is followed few hypo-

tensive reactions will occur. The infusion should not be continued until a *normal* blood pressure is obtained since a further lowering of blood pressure may be noted five to ten minutes after the infusion has been stopped. A marked degree of hypotension might thereby be produced if this method were followed. Pressure should be stabilized at

between 150-170

100-110 The average dose of Hexamethonium

necessary to produce this effect is between 15 to 60 mg., although occasionally 100 to 125 mg. may be necessary.

If a reaction does occur the patient should be maintained in a marked Trendelenburg position until the blood pressure returns to more normal levels. It will rarely be necessary to do anything further but if blood pressure remains exceptionally low, small doses (2.5 to 5 mg.) of intravenous neosynephrine will promptly raise the pressure level. This or any pressor drug should be used with care after Hexamethonium since patients "under blockade" are extremely sensitive to them. All patients should remain recumbent or semi-recumbent for at least one and one-half to two hours after the use of Hexamethonium. After the initial intravenous dose, subsequent doses may be given subcutaneously (Approximately 5 to 15 mg. more than the effective intravenous dose every three to four hours). Utilizing this method we have never seen a patient whose blood pressure could not be lowered and controlled. This, of course, does not imply that recovery will occur, especially if renal disease is severe, but with effective blood pressure control symptomatic relief may be obtained and further cerebral damage curtailed. Hexamethonium or any of its derivatives should be used with care in patients with renal impairment because of the danger of further reducing renal blood flow and increasing the degree of azotemia.

An example of the above method of therapy of hypertensive encephalopathy is provided by the following case.

A forty-four-year-old negro male with known hypertension of sixteen years' duration began to experience severe headaches, nausea and vomiting. His blood pressure varied between 210/130 and 220/150 and fundoscopic examination revealed a Grade IV retinopathy (hemorrhages, exudates, papilledema). Left ventricular hypertrophy was present but renal function was adequate. The patient was considered to be a severe hypertensive with encephalopathy. Twelve milligrams of Hexamethonium were given as described above. His pressure fell and his headache disappeared within twenty minutes. He was maintained on 20 mg. of Hexamethonium subcutaneously on a four-hour schedule for the next twenty-four hours. Symptoms cleared. The patient was then placed on oral Hexamethonium and Apresoline. Headache, nausea and vomiting did not return and papilledema cleared.

An excellent symptomatic and blood pressure response has been obtained in other patients but some have progressed to a fatal outcome. At postmortem, severe renal disease has been found. It is, of course, impossible to do more than alleviate symptoms in these individuals.

The use of Arfonad (a Thiophanium derivative and effective ganglionic blocking agent)^{22b} has been advocated for the treatment of hypertensive crises by some observers. This compound lowers blood pressure but has an extremely short duration of action. It may be administered in an infusion (1 to 2 mg/cc in 5 per cent dextrose) or as a single intravenous injection 1 to 2 mg/kg. Another, more potent, longer acting ganglionic blocking agent, Ansolyse^{107 218} (pentapyrrolidinium) is also an effective blood pressure lowering agent. Although this drug appears to be superior to Hexamethonium when utilized orally in the long term management of the ambulatory hypertensive patient, its use intravenously in hypertensive encephalopathy would not appear to present any advantages over the use of Hexamethonium. It is approximately five times

as potent as Hexamethonium and is administered in doses of 3 to 15 mg. when given parenterally.

If nausea and vomiting prove troublesome during an episode of hypertensive encephalopathy, Thorazine, Dramamine or Marazine should be given immediately. One to 2 cc of Thorazine intramuscularly every four hours, and then 25 mg. every four hours by mouth as soon as this is tolerated, will rarely fail to relieve this annoying and potentially dangerous symptom.

There is no longer any reason to be content with overwhelming these patients with sedatives, and allowing the disease to run its course. Many can be greatly benefitted by therapy. Undoubtedly within the next few years safer and better drugs will be introduced for use in these crises.

Pheochromocytoma.—Pheochromocytoma, a tumor which secretes both epinephrine and norepinephrine, is usually found in the adrenal medulla or the retroperitoneal chromaffin tissue. It may cause attacks of severe hypertension with headaches, palpitations, tachycardia, vertigo, abdominal pain, nausea, vomiting and profuse sweating.⁴⁴⁵ Because of the increased amount of circulating epinephrine in patients with a pheochromocytoma an elevation of the basal metabolic rate sometimes occurs. Occasionally glycosuria and an elevated blood sugar are present as a result of an increased glycogen breakdown secondary to epinephrine action. The attacks of palpitation, sweating, etc., may be sudden in onset, and occasionally can be induced by palpating the region of the kidneys, although in our experience this is unusual. In many cases of pheochromocytoma, the hypertension is persistent and chronic headaches and continuous sweating are present. The diagnosis of this tumor should be considered in hypertensive patients who perspire profusely or who have an elevated basal metabolic rate, a glycosuria or a marked loss of weight. It may be confirmed by the use of one or several specific tests. If the patient is seen during a normotensive phase, the use of

histamine²⁷² (.025 mg. intravenously) will produce a rise in blood pressure and a reproduction of the acute attack. The rise in blood pressure is probably the result of a dilatation of blood vessels in the tumor and/or a direct stimulating effect with a resultant increased outpouring of epinephrine.

Benzodioxane,¹²⁰ and Regitine,⁸⁵ both of which cause a fall in the blood pressure and bring relief of symptoms as a result of their action in blocking the effects of circulating epinephrine or norepinephrine, are to be used during the hypertensive phase in patients with pheochromocytoma. The Regitine test (5 mg intravenously) is a simple, safe office procedure. If this test is positive the results should be checked with a Benzodioxane Test (1 mg/10 lbs given slowly intravenously). Reactions (marked elevation of blood pressure or convulsions) occasionally occur with Benzodioxane when the drug is used in patients with hypertension not secondary to pheochromocytoma, and this test should only be performed in the hospital. "False positive and negative" results have been reported with all of these agents and several tests should be done to check the diagnosis.

Sedation should not be employed for thirty-six hours before the Regitine Test as "False Positive" results are often obtained in patients receiving sedatives. Interpretation of any test in the presence of uremia should be guarded. Assay of urinary excretion of epinephrine and norepinephrine¹¹⁹ may eventually prove to be of more value in the diagnosis of this tumor than any of the above tests.

Although calcification in a pheochromocytoma is uncommon, its presence in the region of the adrenal gland may suggest the diagnosis in a hypertensive patient with any unusual symptoms.²²⁵ The presence of diffuse electrocardiographic changes (probably as a result of epinephrine effect) in a patient with hypertension should also suggest the diagnosis.²⁷⁹

Since hypertension secondary to pheochromocytoma is the only type of hypertension that can almost always be cured by surgical intervention, we feel that *all* patients with either fixed or paroxysmal hypertension should be studied for the presence of this tumor. Although the tumor is relatively uncommon the discovery of only an occasional case is extremely gratifying.

Acute hypertensive episodes secondary to pheochromocytoma should be treated with intravenous and/or oral Regitine. We have also used oral Dibenzylidine (10 mg three times daily) to maintain several patients in a normotensive state while they awaited surgical exploration, although we do not feel that preoperative therapy of this type is necessary in most cases. Hypertensive attacks that occur as the tumor is palpated during laparotomy usually are transient and no therapy is required. If they persist, 5 mg. of intravenous Regitine will control them.

If shock occurs after removal of the tumor this can be controlled successfully by the use of a continuous infusion of norepinephrine (Levophed) (4 mg/1000 cc of glucose in water).

Chapter

7

DISSECTING ANEURYSM

DISSECTING aneurysm of the aorta is relatively uncommon, but its diagnosis is important for two reasons (1) if an erroneous diagnosis of acute coronary occlusion or pulmonary infarction is made and anticoagulant therapy is given, the possibility of recovery from a dissecting aneurysm is further reduced, (2) if a method for the surgical treatment of dissecting aneurysms is perfected, early diagnosis will be essential for success. Only a small percentage of the cases is diagnosed ante-mortem, probably because of the failure to suspect the presence of the condition. Contrary to general belief, a dissecting aneurysm is not invariably fatal, 10 to 25 per cent of patients have lived for from three months to eight years following its onset ¹⁴⁸ The dissection may be acute, lasting only a few hours, and terminate in death as a result of the rupture of the aorta, or it may be subacute, causing repeated bouts of pain for a period of two to five days, and end in recovery.

The attack does not appear to be precipitated by unusual exertion, usually occurs in elderly patients with hypertension, and is sudden in onset. At least two-thirds of cases occur in males ²⁸ In the younger age group dissecting aneurysm is occasionally associated with coarctation of the aorta and pregnancy. Occlusion of a branch of the aorta, i.e., iliac, intercostal, or coronary, is common. There is a sharp, knife-like pain in the chest and/or back, with occasional radiation to the epigastrium, the lower abdomen, the flanks and

the legs. Syncope is noted in 10 per cent of the cases and shock may occur quickly. The patient may writhe in severe pain, but unlike the pain of coronary occlusion this is not "crushing" or "constrictive" and does not require a minute or two to reach its maximum intensity. Various neurologic signs may appear, including paraplegia, which is caused by hemorrhagic infarction of the spinal cord.²⁹ An aortic diastolic murmur is heard in many cases. Electrocardiographic evidence of left ventricular "strain" or coronary insufficiency may appear, or of coronary occlusion if the dissection occludes a coronary artery. A coronary occlusion secondary to a dissecting aneurysm may sometimes be differentiated from the ordinary type of coronary occlusion by the presence of pain in the upper or lower back, the lower abdomen or the legs, by the sudden appearance of a diastolic murmur, or by the presence of neurologic changes, especially "sensory loss" in the lower extremities. If a pleural or pericardial effusion or hemorrhage into the wall of the aortic arch occurs, a widened mediastinum may be detected on fluoroscopy or x-ray. This procedure should be carried out with great caution and actually should not be used if the diagnosis is fairly well established from other findings. The diagnosis should be considered in any patient with hypertension who suddenly develops severe knife-like pain in the chest, back, epigastric region or legs, and in whom a diastolic murmur suddenly appears.

A case in which the correct diagnosis was suggested during life was that of a hypertensive man of forty-two who developed episodes of severe substernal pain and shock with apparent complete recovery between attacks. The electrocardiogram showed the deep Q-waves and RS-T elevations characteristic of coronary occlusion. On the third day, during one of his attacks, the heart sounds suddenly became faint and the area of cardiac dullness increased. A pericardial tap revealed hemopericardium and a diagnosis of dissecting aneurysm was made. He died almost immediately.

and postmortem examination disclosed a dissection involving a coronary artery.

Many times, however, the history and findings are completely atypical and a correct diagnosis cannot be made. For example, A sixty-three-year-old white male complained of sudden, severe, intermittent pain in the left lower quadrant. He had experienced mild pain in that region eight months before for a period of four or five days, but the pain had not recurred until the present admission. Examination revealed an ill-defined mass in the left lower quadrant. His blood pressure was 170/100 and no murmurs were heard. A diagnosis of carcinoma of the colon or diverticulitis was made. Several days later the patient was found to be quite pale and the mass appeared to be larger. Twenty-four hours later he died suddenly. Necropsy showed evidence of dissection of the aorta from the thoracic to the abdominal portion with rupture of the abdominal aorta and retroperitoneal hemorrhage.

When the presence of a dissecting aneurysm is suspected, the patient should be put to bed immediately. Repeated doses of morphine should be given if necessary to attain complete sedation. He should be kept in bed for at least three weeks and then should gradually be permitted to walk. Anticoagulants should not be used. If an accurate diagnosis is made, and if treatment is instituted early, a much larger percentage of cures may be effected. Since the pain pattern in many cases of dissecting aneurysm may be intermittent, with one or many hours of "quiescence" in between periods of pain, the prognosis should be guarded until at least one week after all symptoms have disappeared.

In view of the tremendous advances in vascular surgery it is not unlikely that a procedure will be developed to rechannel the blood in the dissected coats of the aorta into the lumen of the vessel. A prerequisite to this, however, is the discovery of methods to detect the site and path of the dissection.

Chapter

8

TRAUMATIC HEART DISEASE

THE heart may be injured directly by penetrating bullet or knife wounds or indirectly by severe blows to the chest^{11,18}

The diagnosis of a *penetrating wound of the heart* is simple if external evidence of the injury is present. Occasionally, however, a bullet or a needle which had entered the body at some remote point may be carried to the heart through the veins¹⁹ and produce cardiac damage. In these cases the diagnosis is often difficult. If a patient with a recent penetrating injury of the chest wall presents evidence of blood loss, congestive heart failure or shock, trauma of the heart should be suspected. The most common result of such trauma is hemorrhage into the pericardial sac or laceration of the myocardium with infarction. In rare instances valvular rupture may occur. When hemo-pericardium develops, fluoroscopy or x-ray may show evidence of a widened cardiac shadow with diminished pulsations or the electrocardiogram alone may show characteristic RS-T elevations. Occasionally findings in such cases are similar to those seen in myocardial infarction. If there is evidence of increasing hemo-pericardium and cardiac tamponade, *i.e.*, shock, with a weak thready pulse and drop in blood pressure (low cardiac output), or congestive heart failure, a pericardial tap should be performed immediately and as much blood as possible aspirated^{20,4}. If blood reaccumulates, or if the symptoms persist or recur, a severe

myocardial laceration has occurred and surgical repair should be carried out immediately

Shock should be treated as efficiently as possible before and during the operation. The Trendelenburg position, frequent transfusions of blood (plasma or a volume expander such as dextran if blood is not available), and the use of morphine or Demerol for pain and apprehension often help the patient to surmount the crisis until the wound is repaired. Despite the presence of cardiac tamponade the cardiac output may be increased and the patient's condition improved by these measures.¹⁴²

Excellent reviews of the technique of repairing cardiac lacerations or removing foreign bodies from the heart have been written.^{18,132} These surgical procedures need not be discussed here. Complications such as cardiac arrest encountered during operation are discussed in Chapter 9. In experienced hands, the operative mortality rate is 25 to 30 per cent.³¹ The site repaired usually heals and a firm scar develops. Despite the fact that a major coronary artery is often ligated, infarction does not always occur, and even if it does, cardiac function is usually good.¹⁴² A case seen by us demonstrated this finding. W.C., a thirty-five-year-old laborer was stabbed in the region of the heart with a long knife two hours prior to admission. There was profuse hemorrhage but the patient walked several blocks to the hospital. He complained of pain in the chest but was neither dyspneic nor cyanotic. The blood pressure was 100/80. Heart sounds were distant and a pericardial rub was heard which soon disappeared. An x-ray of the chest and an electrocardiogram were negative. In view of the profuse bleeding, an exploratory operation was performed. A laceration of the parietal pericardium, with a moderate hemopericardium and an insignificant stab wound of the anterior surface of the heart were found. Upon gentle manipulation the occluding blood clot was dislodged and free bleeding occurred, this could be controlled only by the insertion of

three sutures, two of which entered the chamber. Following the operation, the electrocardiogram showed RS-T elevations followed by T-wave inversions in the standard leads (chest leads were not taken because of the chest strapping), i.e., the typical serial changes of acute pericarditis. In addition, a Q-wave appeared in lead II, this produced a W-shaped QRS which was still present a year later when the T-waves in the standard leads had returned to normal. The alteration in the QRS complex was probably produced by ligation of a coronary artery during the operation, but the patient made an excellent functional recovery.

In some cases a bullet or shrapnel fragment may penetrate the chest and lodge in the heart without producing any cardiac symptoms. The fragment may be visualized by x-ray or fluoroscopy. It is best not to remove the foreign body unless a pericardial reaction occurs. We have observed two cases of interventricular septal defect secondary to bullet wounds where the fragment had penetrated the septum from left to right and lodged in the right ventricle. Both patients did well without surgical removal of the bullet.

Nonpenetrating chest injuries may produce myocardial contusion with signs and symptoms that are difficult to distinguish from those noted in spontaneous coronary occlusion or coronary insufficiency. The causal relationship between trauma and myocardial damage is frequently difficult to establish. As a rule, only a very severe blow to the chest wall produces a detectable cardiac injury. Many boxers and victims of accidents who sustain injuries to the chest show no anatomical or electrocardiographic evidence of myocardial damage.^{53 123}

Many cases of alleged traumatic coronary occlusion reported in the literature probably represent instances of coincidental occurrence of coronary occlusion and trauma. The electrocardiograms which have been reported in a large number of these patients as showing an occlusion

actually show only ST and T-wave changes which occur in coronary insufficiency and not in occlusion^{234 224} In some instances a careful history will elicit the fact that the precordial pain preceded the accident. Although we are certain that episodes of coronary insufficiency may be precipitated by effort or trauma, we believe that the occurrence of coronary occlusion is coincidental with extreme physical exertion and that the occlusion had occurred prior to the injury^{208 209}

Trauma to the chest wall occasionally may produce sub-endocardial tears and myocardial contusions in otherwise normal hearts²⁴³ All cases of so-called "steering wheel" accidents should be carefully observed for several days in a search for electrocardiographic or clinical evidence of heart damage, even in the absence of rib fracture or severe external injury²⁴⁰ These patients may show various arrhythmias without other changes for several days following the accident. We recently observed a forty-year-old man with a persistent rapid atrial fibrillation for over two weeks following a severe "steering wheel" accident. Hemopericardium was present and drug therapy was ineffective in controlling the arrhythmia. Fortunately, this eventually subsided spontaneously. All cases with a definite history of severe chest trauma that develop precordial pain or discomfort at the time of the injury should be suspected of having sustained a myocardial contusion, the patients should, therefore, be put to bed and given adequate sedation.

The following criteria are helpful in determining whether the cardiac damage was caused by a specific trauma²⁴⁰ The decision must be made on the basis of the facts in each case, but, in general, we require the presence of two or more of these criteria for causal relationship.

1. Significant electrocardiographic changes must be present.
2. Cardiac enlargement unexplained by pre-existent cardiac disease or hypertension must appear.

- 3 Abnormal rhythms (excluding premature beats unless they are accompanied by other electrocardiographic changes) must occur
- 4 A pericardial friction rub must be heard, or evidence of effusion found
- 5 Congestive heart failure, precipitated by the blow or strain, must develop
- Clinical signs and laboratory evidence of coronary insufficiency and/or myocardial infarction must become manifest within twenty-four hours following the incident

Patients with myocardial contusion should be treated in the same manner as those with nontraumatic coronary insufficiency or occlusion with the exception that anticoagulant drugs should be withheld because of the frequent occurrence of pericardial hemorrhage. If hemopericardium does occur, the blood should be repeatedly aspirated if necessary.

Rupture of the heart occurs in a few of these cases within ten days to two weeks after the trauma, but if the patient is put to bed as soon as symptoms appear, the incidence of this fatal complication may be reduced.

Many cases of traumatic myocardial damage recover completely after three to five weeks of bed rest and show no permanent clinical or electrocardiographic evidence of heart disease.

Chapter

9

SURGICAL EMERGENCIES IN CARDIO-VASCULAR DISEASE THEIR PREVENTION AND TREATMENT

Surgery in the Cardiac Patient.—Recent advances in surgical techniques and anesthetic management have significantly reduced the mortality and morbidity associated with surgery in the patient with heart disease. The necessity for operative intervention, however, in individuals with cardiovascular disorders has also greatly increased with the increase in the aged population, and there is an even greater need for the careful medical supervision of such patients in order to prevent the development of postoperative acute congestive failure, coronary insufficiency, tachycardias and pulmonary embolism.

Most patients with heart disease tolerate an operation well, the exceptions being those who are in intractable failure or are recovering from a recent myocardial infarction. Serious complications may not develop even in these patients if the pre- and postoperative treatment is properly administered.

Age should not be a deterring factor in patients who require surgery.²³² In fact, many elderly patients with heart disease and severe cardiac disability are occasionally improved by the surgical removal of a "focus of irritation." For example: M S., a fifty-nine-year-old housewife, who had been an invalid because of severe recurrent attacks

of angina pectoris, also experienced frequent biliary colic. The surgeons were hesitant about operating on her because of her cardiac condition but they were finally prevailed upon to do so. She tolerated the operation well and has remained practically free of her anginal attacks for many years. Another example was M R, a sixty-year-old woman with gall bladder disease who suffered an acute posterior wall infarction. Her convalescence was marked by repeated attacks of severe biliary colic. A cholecystectomy was performed one month after the occlusion had occurred. Her post-operative course was uneventful and since then she has not complained of pain. It is usually advisable to wait two to three months after the electrocardiogram has stabilized following a myocardial infarction before surgery is performed, unless, of course, an acute situation makes immediate intervention a necessity.

In the presence of *heart failure* operation should be performed only when it is urgently needed and only after careful preparation if time permits. Even when proper preparation is not possible, the results have often been good. When congestive failure exists, rapid intravenous digitalization should be carried out and mercurial diuretics given. In patients with rheumatic heart disease and atrial fibrillation the rate should be slowed by intravenous digitalis therapy before surgery is attempted. Persons with compensated heart disease may be expected to withstand operative procedures as well as those whose hearts are normal if certain precautions are taken. Special attention should be paid to the amount of fluids and sodium given pre- and postoperatively, and an adequate quantity of oxygen should be administered during the operation. The administration of large quantities of intravenous saline after an operative procedure should be avoided in cardiac patients in order to prevent pulmonary edema and congestive heart failure.

In patients with coronary artery disease particular care should be exercised to avoid hypoxia, especially during the induction of anesthesia, and *hypotension* during the course of the operation. Spinal anesthesia has usually been considered dangerous for patients with coronary artery disease or severe hypertension, but we have found it effective and safe for operations on the lower abdomen or pelvis. Intramuscular methoxamine (Vasoxyl), neosynephrine or ephedrine may be given prophylactically to prevent a precipitous fall in blood pressure during spinal anesthesia and intravenous Vasoxyl (5 mg) or neosynephrine (.5 to 10 mg) should be available for immediate use if hypotension should occur. Norepinephrin (Levophed), 4 mg in 1000 cc. of glucose, is also effective in maintaining blood pressure during surgery if hypotension is not secondary to blood loss. In the latter case blood should be given as soon as possible.

Although it was formerly believed that local or regional anesthetics were to be preferred in patients with coronary artery disease, experience has demonstrated that general anesthetics are more satisfactory, provided that adequate oxygenation and ventilation are maintained at all times. Apprehension and some degree of pain which are often associated with local anesthetics are thereby eliminated. If a general anesthetic is used, ether is preferred or a thiopental induction followed by oxygen and cyclopropane may be used. Since the latter may induce ventricular arrhythmias,¹⁶¹ quinidine, two doses of 6 gr. (4 gm), or Pronestyl¹⁶² (10 to 15 gm) should be given orally before the operation, particularly in patients with a history of arrhythmias. These agents markedly reduce the incidence of tachycardias that occur during surgical procedures. If atrial tachycardia, flutter or fibrillation develops, quinidine should be given intramuscularly. If a ventricular arrhythmia sets in despite these prophylactic measures, Pronestyl may be injected intravenously, but slowly and cautiously with electrocardiographic control.

The incidence of postoperative thromboembolism and pulmonary infarction is higher in cardiac than in noncardiac patients and, in fact, probably accounts for a higher percentage of deaths in these patients than any other single complication. The most important factor in preventing these complications is early ambulation, particularly in the obese. The patient should also be instructed to turn in bed as soon as possible, to move his legs within several hours after the operation, and to avoid straining at stool. Should pulmonary infarction occur, despite these measures treatment is given as outlined in Chapter 3.

During the course of cardiac surgery for the correction of either congenital or acquired valvular defects many unusual arrhythmias may occur, especially during the period of valve manipulation or "fracture." The majority of these arrhythmias are supraventricular and cease spontaneously without the use of drugs, the incidence of these arrhythmias cannot be reduced by the use of preoperative Pronestyl or quinidine. If frequent ventricular premature beats or aberrant ventricular beats occur, or if ventricular tachycardia is noted during heart manipulation, the surgeon should immediately stop all surgery for a short time and the anesthesiologist should lighten the depth of anesthesia and increase the ventilatory exchange as well as oxygenation. It is equally as important to remove excess carbon dioxide as to provide adequate oxygen. Usually ventricular arrhythmias that occur during cardiac surgery will respond without further therapy. The situation in these instances, where the arrhythmia is due to direct manipulation of the heart, is different from those in which surgery in a remote portion of the body is being performed. If ventricular tachycardia persists after manipulation has been stopped, intravenous Pronestyl should be given. If cardiac arrest or ventricular fibrillation occurs, it should be treated as outlined below.

Cardiac Arrest.—Cardiac arrest may occur during the course of an operative procedure in both normal and cardiac patients. In most instances it is due to an overdose of anesthetic agent. However, it may be caused by marked vagal stimulation during the operation and can be prevented in some cases by the intravenous administration of atropine, 1 mg. (1/100 gr.), sixty minutes before surgery, or 3 mg. (1/150 gr.) intramuscularly thirty minutes prior to operation.¹⁶² Cardiac arrest occurs in operations on the chest more often than in abdominal or pelvic surgery. When it does occur, the instantaneous execution of a well-planned course of treatment is essential to save the patient's life.¹⁶⁰

A sixteen-year-old boy, who was operated upon for atelectasis of the left lung secondary to fibrosarcoma of the bronchus, furnishes a dramatic example of the success of cardiac resuscitation, even after a forty-minute period of cardiac arrest.* He received cyclopropane and ether at 8:10 A.M., at 8:25 A.M., an oral endotracheal tube was introduced. At 8:27, the left hemithorax was entered. Sixty mg. of procaine hydrochloride were injected into the tubing of an intravenous drip. The pulmonary artery was ligated at 9:05, when cardiac arrest occurred. Artificial respiration was begun immediately by intermittent manual compression of the breathing bag of the anesthetic apparatus. Simultaneously, manual compression of the heart was started at a rate of 50 per minute. At 9:10, a mixture of 80 mg. of procaine and 1 mg. of epinephrine was injected into the left ventricular cavity. At 9:25, 60 mg. of procaine and 1 mg. of epinephrine were given, and at 9:30, 80 mg. more of procaine were injected. Cardiac action did not return. At 9:45, the pericardium was grasped with a hemostat, preparatory to incision. At that moment the heart suddenly beat violently and normal pulsation and respiration began. The pneumonectomy was completed without mishap. On the

*This case is described with the permission of the operating surgeon, Dr. Arthur E. W. Touroff.³¹³

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If respirations have ceased, artificial respiration should be started simultaneously with the exposure of the heart. If possible, an endotracheal tube should be inserted and the lungs inflated and deflated by mechanical means. This is by far the most efficient method of supplying oxygen.⁶⁹ If endotracheal tubes are not available, however, the resumption of respiration may be effected by mouth to mouth breathing, alternating manual pressure and release of the costal cage, or the use of a tight-fitting face mask, pressure being applied intermittently to the breathing bag. When the latter method is used there is real danger of forcing air into the stomach and causing marked gastric dilatation.⁴⁴⁰ One hundred per cent oxygen should be given and CO₂ mixtures should be avoided since the patient already has accumulated an excessive amount of CO₂ in the tissues. Adequate oxygenation of the brain and other organs may be maintained by use of any one of the above procedures until spontaneous respiration and circulation are resumed.

When hemorrhage has occurred and the blood volume is markedly decreased, intra-arterial transfusions of blood should be given at the rate of 150 to 200 cc/minute. These transfusions are given at a pressure higher than the systolic blood pressure.

In many cases, patients who had apparently expired will be completely revived and will recover with no ill effects if the suggested treatment is undertaken promptly. If the surgeon is properly equipped and psychologically ready for this emergency, good results may be expected. Unfortunately, there is often a delay before adequate therapy is started, and although the patient survives, extensive cerebral damage from prolonged anoxia has occurred and the patient remains a neuropsychiatric cripple.¹⁰⁹

Resuscitation of patients who develop cardiac arrest while on the wards or at home is rarely successful because of the delays that are of necessity encountered in these situations. Our own experience has indicated that cardiac compression

should never be attempted except under operating room conditions where speedy action is possible. In other situations the use of intracardiac Adrenalin or vigorous thumping on the chest as outlined in Chapter 1 will produce results comparable to those obtained by "slashing open" the chest and massaging the heart.

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161

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INDEX

Bold face numbers indicate principal reference

A

ABDOMINAL belt for postural hypotension, 119
 distention *See* Distention
 pain in digitalis toxicity, 25
 in pheochromocytoma, 136
 in quinidine therapy, 20
Acetylcholine, in atrial tachycardia, 40
Acetyl-strophanthidin, 29
Acidosis, in congestive failure 68
ACTH, potassium loss, 124
 quinidine sensitivity, 21
 rheumatic fever, 123, 124
 sodium retention, 124
Addison's disease, postural hypotension, 119
Adrenal cortical hormones, in rheumatic fever, 123
Adrenalin *See* Epinephrine
Adrenergic blocking agents, 47
Agranulocytosis with Pronestyl, 45
Alcohol, in angina pectoris, 80
 in atrial tachycardia, 13, 35
 in paravertebral block, 82
 in premature beats, 52
 in salt poor diet, 65
Alcohol vapor, in pulmonary edema, 62
Allergy in, atrial tachycardia, 13, 35
 to Mechohyl, 40
 to quinidine, 20
Alpha Tocopherol (Vitamin E), in angina pectoris, 79
Ambulation, in coronary occlusion, 112

Ambulation in surgical emergencies, 150
Aminophyllin, aluminum hydroxide mixtures, 79
 in cerebral embolization, 108
 in Cheyne-Stokes respiration 60
 in congestive failure, 69
 in coronary occlusion, 94
 in emergency kit, II
 in nocturnal dyspnea, 58
 in pulmonary edema, 60
 suppositories in coronary insufficiency, 86
Ammonium chloride, cause of hypochloremic acidosis, 69
 electrolyte disturbance 68
 in congestive failure, 68
 in hypochloremic alkalosis 69
Amyl nitrite, danger of syncope 119
 in hiccup, 74
Amytal sodium, coronary occlusion, 94
Anemia, heart failure, 57, 72
 high cardiac output, 55, 63
 syncope, 122
Anesthesia, cyclopropane, 149
 ether, 149
 in coronary insufficiency, 68
 in coronary occlusion, 110
 in surgical procedures, 148, 149
 local, 149
 oxygen, 148
 spinal, 88
 thiopental, 149
Aneurysm, arteriovenous, 72

- Aneurysm, dissecting, 139-141
 ventricular, 111
 Angina decubitus, 80
 therapy, 81-83
 Angina pectoris, 70-83
 anxiety, 78
 biliary colic, 76
 cold, 77
 coronary insufficiency, 76
 differential diagnosis, 76
 effort, 77
 excitement 77
 in hyperthyroidism, 81, 82
 in surgical emergencies, 148
 myocardial necrosis absent, 77
 preceding coronary occlusion, 77
 "status angiosus," 78, 80
 subendocardial ischemia, 77
 surgical procedures, 82, 83
 anastomosis of aorta to coronary sinus, 83
 cardiac vein ligation, 81
 cardiopercutaneous, 83
 implantation of internal mammary artery, 83
 posterior rhizotomy, 82
 thoracic sympathectomy, 82
 treatment, alcohol, 80
 Alpha Tocopherol (Vitamin E), 79
 aminophylline-aluminum hydroxide mixtures, 79
 antithyroid drugs, 81
 heparin, 79
 hygiene, 79
 ins, 81
 Kheslin, 79
 Vitamin E, 79
 nitrites, 78
 nitroglycerin, 77
 Nitroglyn, 79
 papaverine, 79
 paravertebral block, 82
 Parental Phosphate, 79
 Angina pectoris, treatment, Peritrate, 78
 quinidine, 79
 Reserpine, 79
 sedatives, 78
 testosterone, 79
 vitamin E, 79
 whiskey, 80
 Ansolexin in hypertensive encephalopathy, 135
 Anticoagulant therapy, 85, 92, 101, 105-109
 clotting time, 105
 danger in acute pericarditis, 92, 107
 danger in dissecting aneurysm, 141
 danger in traumatic heart disease, 148
 drugs, Depo heparin, 105
 Dicumarol, 105, 108, 109
 Hedulin, 105, 108
 heparin, 105
 protamine sulphate, 105
 transfusion, 107
 Tromexan, 105, 108
 vitamin K, 106, 107
 hemorrhage, 105, 107
 pericardial, 107
 indications, cerebral embolization, 108, 127
 congestive failure, 84, 101
 coronary occlusion, 101
 peripheral arterial embolization, 108, 127
 phlebitis, 101
 pulmonary embolism, 101
 rheumatic heart disease 127
 shock, 101
 oral, 108
 protrombin time, 105-107
 urinalysis, 106
 Antidiuretic drugs, 70
 hormone in heart failure, 54
 Antifoaming agents in pulmonary edema 82

- Antihistamines, in quinidine sensitivity, 21
- Anti-hypertensive drugs, inducing coronary insufficiency, 85
in hypertensive encephalopathy, 132
- Antithyroid drugs, angina pectoris, 81
- Anxiety, in angina pectoris, 80
in arrhythmias, 11
in coronary occlusion, 91
in premature beats, 52
in syncope, 120
- Aortic stenosis, calcific, syncope 121
coronary insufficiency as a result of, 85
valvulotomy, 122
- Apresoline in hypertensive encephalopathy, 133
- Arfonad in hypertensive encephalopathy, 135
- Arrhythmias, 11-53
angina, 11
anxiety, 11
collapse, 11
convulsions, 12
heart failure, 11, 22
in coronary occlusion, 13, 97
in Graves' disease, 14, 18, 29, 30, 35
rheumatic fever, 18, 29, 31
shock, 12
surgical emergencies, 149-150
syncope, 11, 12, 51
traumatic heart disease, 145
treatment, atropine, 28, 40, 49
Cedilanid, 23, 29, 36
dibenzamine hydrochloride, 47
digitalis 14, 18, 19, 22, 24, 25, 29-33, 38, 42, 47, 97
digitalis leaf, 24
digitoxin, 23, 25
digoxin, 23, 25
magnesium sulfate, 46
ouabain, 22
- Arrhythmias, treatment, potassium chloride, 22, 28, 46, 49
Pronestyl, 22, 38, 39, 43-46
47, 49, 97
propyl thiouracil, 30
quinidine, 14, 16, 17, 18, 20, 21, 22, 32, 33, 36, 38, 43, 47, 49, 97
strophanthum, 22
vertigo, 11
vomiting, 11
- Arteriosclerotic heart disease, atrial fibrillation, 14
- Arterio-venous fistulae, 55, 57, 72
- Arthritis, in rheumatic fever, 123
- Artificial heart, in cardiac surgery, 131
respiration, in surgical emergencies, 151, 153
- Asites, in heart failure, 57, 73
in rheumatic heart disease, 73
- Asthma, bronchial, coronary insufficiency, 86
danger of Mecholyl, 40
- Asthma, cardiac, 57 See Cardiac asthma
- Aystole See Cardiac standstill
- Atabrine, in ventricular tachycardia, 46
- Atrial fibrillation, 11, 12, 14-31
alcohol, 13
A-V block, 21, 23, 28
caffeine, 13
chronic, 19
differential diagnosis, Table 1, 15, 41
due to Mecholyl, 40
embolization, 19
emotional disturbance, 13
heart failure, 14, 22
in arteriosclerotic heart disease, 14
in coronary occlusion, 97
in hyperthyroidism, 13, 14, 29, 30, 31
in metastatic malignancy, 31
in mitral stenosis, 126
in pneumonia, 14

- Atrial fibrillation, in rheumatic fever, 29, 31
 in rheumatic heart disease, 126
 in surgical procedures, 14, 149
 in traumatic heart disease, 145
 mechanism of circus movement, 14
 ectopic focus (unitary theory), 14
 paroxysmal, 14
 pulse deficit, 14
 sedation, 14
 sinus rhythm restoration, 19
 tobacco, 13
 treatment Cedilanid, 23
 digitalis, 14, 19, 22-25, 31
 digitoxin, 23
 digoxin, 23, 24
 gitalin, 23, 24
 ouabain, 22, 23
 potassium salts, 22, 28
 Pronestyl, 22
 quinidine, 16-20
 strophanthin ("Strophosid"), 22, 23
 valvulotomy, 129
- Atrial flutter, 11, 12, 31-35
 alcohol, 13
 A-V block, 31
 bundle branch block, 31
 carotid sinus pressure, 31
 chronic, 32
 differential diagnosis, Table, 15
 due to ectopic focus, 14
 esophageal leads, 32
 in coronary occlusion, 97
 in surgical emergencies, 149
 tobacco, 13
 treatment, digitalis, 32, 33
 Pronestyl, Table, 41
 quinidine, 32, 33
 venous pulsations, 31
- Atrial tachycardia, 11, 12, 35-42
 alcohol, 13, 35
 Atrial tachycardia, allergy, 13, 35
 A-V block, Figures, 34, 35
 cardiac output, 11
 carotid sinus, 35-36
 differential diagnosis, Tables, 15, 35, 41
 electrocardiogram, 12
 emotional disturbances, 35
 gastric distention, 35
 heart failure, 58
 in coronary occlusion, 69
 in Graves' disease, 35
 in infection, 35, 38
 in surgical emergencies, 49
 pulmonary edema, 6
 syncope, 12
 tobacco, 13, 35
 treatment, acetylcholine, 40
 Cedilanid, 36
 digitalis, 38
 eyeball pressure, 36
 ippecac, 39
 Mecholyl, 36, 40
 neosynephrine, 39
 Pronestyl, 38, 39
 quinidine, 36, 38
 vagus stimulation, 35, 39
 Vasoxyl, 39
 vomiting, induction, 39
- Atrial thrombus, in mitral stenosis, 126
 waves, in atrial flutter, 31
 in esophageal leads, 32
- Atropine, A-V block, 28
 cardiac arrest, 151
 in emergency kit, 10
 in postural hypotension, 119
 in pulmonary edema, 59
 in sinus bradycardia, 52
 in syncope, 118
 in ventricular fibrillation, 49
 vs Mecholyl, 36, 40
 vs parasympathomimetic drugs, 40
- Auricular appendectomy, left, 127
- A-V block, 49-51
 digitalis, 25, 68

- Antihistamines, in quinidine sensitivity, 21
- Anti-hypertensive drugs, inducing coronary insufficiency, 85
- in hypertensive encephalopathy, 132
- Antithyroid drugs, angina pectoris, 81
- Anxiety, in angina pectoris, 80
- in arrhythmias, 11
- in coronary occlusion, 94
- in premature beats, 52
- in syncope, 120
- Aortic stenosis, calcific, syncope 121
- coronary insufficiency as a result of 85
- valvulotomy, 122
- Apresoline in hypertensive encephalopathy, 133
- Arfonad in hypertensive encephalopathy, 135
- Arrhythmias, 11-53
- angina, 11
- anxiety, 11
- collapse, 11
- convulsions, 12
- heart failure, 11, 22
- in coronary occlusion, 13, 97
- in Graves' disease, 14, 18, 29, 30, 35
- rheumatic fever, 18, 29, 31
- shock, 12
- surgical emergencies, 149-150
- syncope, 11, 12, 51
- traumatic heart disease, 145
- treatment, atropine, 28, 40, 49
- Cedilanid, 23, 29, 30
- dibenzamine hydrochloride, 47
- digitalis, 14, 18, 19, 23, 24, 25, 29-33, 38, 42, 47, 97
- digitalis leaf, 24
- digitoxin, 23, 25
- digoxin, 23, 25
- magnesium sulfate, 40
- ouabain, 22
- Arrhythmias, treatment, potassium chloride, 22, 28, 40, 49
- Pronestyl, 29, 38, 39, 43-46, 47, 49, 97
- propyl thiouracil, 30
- quinidine, 14, 16, 17, 18, 20, 21, 22, 32, 33, 36, 38, 43, 47, 49, 97
- strophanthum, 22
- vertigo, 11
- vomiting, 11
- Arteriosclerotic heart disease, atrial fibrillation, 14
- Arterio-venous fistulae, 55, 57, 72
- Arthritis, in rheumatic fever, 123
- Artificial heart, in cardiac surgery, 131
- respiration, in surgical emergencies, 151, 153
- Ascites, in heart failure, 57, 73
- in rheumatic heart disease, 73
- Asthma, bronchial, coronary insufficiency, 86
- danger of Mechohyl, 40
- Asthma, cardiac, 57 See Cardiac asthma
- Astysole See Cardiac standstill
- Atabrine, in ventricular tachycardia, 46
- Atrial fibrillation, 11, 12, 14-31
- alcohol, 13
- A-V block, 21, 25, 28
- caffeine, 13
- chronic, 19
- differential diagnosis, Tables, 15, 41
- due to Mechohyl, 40
- embolization, 19
- emotional disturbance, 13
- heart failure, 14, 22
- in arteriosclerotic heart disease, 14
- in coronary occlusion, 97
- in hyperthyroidism, 13, 14, 29, 30, 31
- in metastatic malignancy, 31
- in mitral stenosis, 120
- in pneumonia, 14

- Cardiac arrest, in surgical emergencies, 150, 151-154
in traumatic heart disease, 143
mitral valvulotomy, 130
Procaine, 152
- Cardiac asthma, 57, 58
aminophylline, 58
blood volume, 57
digitalis, 58
diuretics, 58
low sodium diet, 58
morphine, 58
phlebotomy, 58
- Cardiac catheterization, Pronestyl, 43
ventricular arrhythmia, 42
- Cardiac curiosis, in congestive failure, 73
in rheumatic heart disease, 73
- Cardiac compression, in surgical emergencies, 152, 153
- Cardiac enlargement, in traumatic heart disease, 145
- Cardiac failure See Heart failure
- Cardiac massage in cardiac arrest, 151
- Cardiac output, diminished, 55, high, 55
effect of heart rate, 11
in congestive failure, 55
in paroxysmal tachycardia, 11
in postural hypotension, 118
in syncope, 113
in traumatic heart disease, 142
phlebotomy, 55
salt restriction, 55
- Cardiac resuscitation, in surgical emergencies, 151
- Cardiac standstill, 50
atropine, 52
complete A-V block, 50
ephedrine, 50, 52
epinephrine, 50
- Cardiac standstill, in syncope 50
Isuprel, 50
neosynephrine, 45
Pacemaker, 51
Paredrine, 50
Pronestyl, 45
sinus bradycardia, 52
Stokes-Adams seizures, 50
ventricular fibrillation, 49
- Cardiac surgery See Mitral valvulotomy
artificial heart, 131
cross-circulation, 131
hypothermia, 131
- Cardiac tamponade, in rheumatic pericardial effusion, 128
in traumatic heart disease, 142
- Cardiopneumostomy, in angina, 83
- Cardio-pulmonary insufficiency
acute, 62-63, 64
cor pulmonale, chronic, 62, 64
emphysema, 62
heart failure, 63
hematocrit, 63
pulmonary disease, chronic, 62
fibrosis, 64
infarction, 61
infection, 64
respiratory acidosis, 63
right ventricular hypertrophy, 62
treatment, antibiotics, 63
bronchodilators, vaporized, 63
chloral hydrate, 63
digitalis, 63
morphine, 63
oxygen, 63
paraldehyde, 63
phlebotomy, 63
respirators, 63
Vaponephrin, 63
- Carotid sinus, external arteriosclerosis, 118

- Carotid sinus, effect of drugs on,
36
atropine, 36
Benzedrine, 36
epinephrine, 36
lanatoside C, 36
Mecholyl, 36
neosynephrine, 36
quinidine, 36
excision, 118
pressure on, 12, 31, 35, 36
38
convulsions, 36, 118
hemiplegia, 36
supraventricular tachycar-
dia, 35-36, 38
syncope, 36, 115
- Cascara, 74
- Cation exchange resins, 71
constipation, 72
electrolyte disturbance, 71
mercurial diuretics, 71
renal function, 71
- Cedilanid, 28
carotid sinus, 36
in arrhythmias, 23, 29
in atrial and nodal tachycardia
36, 38
in coronary occlusion, 95
in emergency kit, 10
in pulmonary edema, 60, 81
intravenous, 95
test for digitalization, 29
- Cerebral, anoxia, 12, 113
arteriosclerosis, 113
syncope, 113, 122
- Chloral hydrate, acute cardio-
pulmonary insufficiency, 63
- Cholecystitis, causing coronary
insufficiency, 85
- Clotting time, in anticoagulant
therapy, 105
- Coarctation of the aorta, 139
dissecting aneurysm, 139
- Coffee, paroxysmal tachycardia,
13
- Coma, in hypertensive enceph-
alopathy, 132
- Cominode, in coronary occlusion,
111
- Complete A-V block *See* A-V
block
- Congestive failure *See* Heart
failure
- Convulsions, in hypertensive en-
cephalopathy, 132
in syncope, 118, 120
- Coronary insufficiency, 76, 83-89,
147
angina pectoris, 76
diabetes, 89
differential diagnosis, 76
coronary occlusion, 76, 84
electrocardiogram, 84, 145
etiology, anesthesia, 88
aortic stenosis, 85
bronchial asthma, 85
cholecystitis, 85
effort, 85, 88
emotional disturbance, 88
gastro-enteritis, 85
gastro-intestinal hemor-
rhage, 88
heart failure, 85
hemorrhage, 85, Figure,
87
hypertensive crises, 85
hypoglycemia, 89
hypotension, 85
infections, 85
pulmonary embolism, 85,
104
sexual intercourse, 88
shock, 88
spinal anesthesia, 88, 89
surgical procedures, 85, 88
tachycardia, 12, 85
tobacco, 88
traumatic heart disease,
145
- Fever, 84
- Leucocytosis, 86
- Sedimentation rate, 84
- Subendocardial ischemia, 84
subendocardial necrosis, 84

- Coronary insufficiency, treatment,
 aminophylline, 88
 anticoagulants, 85
 Dicumarol, 85
 Dilaudid, 80
 neosynephrine, spinal an-
 esthesia, 89
 nitroglycerin, 85
 transfusions, 88, 89
- Coronary occlusion, 89-112
 ambulation, 112
 anesthesia, 110
 arrhythmias, 92, 97
 arterial embolism, 108
 A-V block, 99
 bed rest, 110
 bundle branch block, 78
 bed-pan, 111
 commode, 111
 cough, 104
 differential diagnosis, 89.
 Tables, 92, 93
 coronary insufficiency, 76,
 84, 92, 93
 dissecting aneurysm, 139,
 140
 pericarditis 92, 93, 107
 distention, 92, 112
 electrocardiogram, 89-91,
 Figure, 91
 embolization, 91, 109
 embryocardia, 89
 fever, 91, 105
 friction rub, 91, 107
 gallop rhythm, 89
 heart failure, 73 89 94-96,
 111
 hemoptysis, 104
 hiccups, 112
 leukocytosis, 105
 mural thrombi, 91
 nausea, 112
 perspiration, 89
 premature beats, 97
 premonitory phase, 85 89
 Figure, 90
 prothrombin time, 105
 pulmonary edema, 78, 89, 96
- Coronary occlusion, pulmonary
 embolism and infarction
 101-105
 sedimentation rate, 91
 shock, 89, 94, 95
 subintimal hemorrhage, 89
 surgical emergencies, 148
 syncope, 121
 tachycardia, Figure, 44
 traumatic, 144
 treatment, aminophylline
 94, 109
 amylal sodium, 94
 anticoagulant therapy, 85
 92, 101-108
 caffeine sodium benzoate
 109
 Demerol, 92
 Dipo heparin, 105
 dibenzylamine, 109
 Dicumarol, 103, 108
 digitalis, 96, 97
 Dilaudid, 93
 diuretics, mercurial, 96
 epinephrine, 94
 heparin, 105
 heparinization, 109
 hospital vs. home care, 94
 Letrophed, 93
 Metipon, 93
 morphine, 92, 93
 neosynephrine, 85
 nitroglycerin, 93
 norepinephrine, 95
 ouabain, 96
 oxygen, 93, 95, 109
 Pantopon, 93
 plasma, 94
 Priscoline, 109
 Pronestyl, 97
 quinidine, 97, 98
 salt restriction, 96
 strophanthum, 96
 transfusion, 94
 Trendelenburg position,
 95
 Tromexan, 105
 vasovial, 95

- Coronary occlusion, vasopressor drugs, 94
 Wyamine, 95
 urinalysis, 106
 ventricular aneurysm, 111
 ventricular fibrillation, 47
 ventricular tachycardia, 97
 vomiting, 92, 112
- Coronary sinus, anastomosis to aorta, 83
- Cor pulmonale, acute See Cardio-pulmonary insufficiency and Pulmonary embolism
 chronic, cardiac failure, 62
 high cardiac output, 55
- Cortisone, in rheumatic fever, 123, 124
- Cough, in coronary occlusion, 104
 in pulmonary embolism, 104
 syncope, 113, 120
- Cross-circulation in cardiac surgery, 131
- Cumethilin, 67
- Cyanosis, in mitral stenosis, 128
 in pulmonary edema, 58
- Cyclopropane, causing arrhythmias, 47, 149

D

- DEFIBRILLATION, in cardiac arrest, 152
- Dehydration, in congestive failure, 64, 66-70
- Demerol, antidiuretic effect, 70
 in coronary occlusion, 92
 in emergency kit, 10
 in pulmonary edema, 59
- Depo heparin, 105
- Deoxycorticosterone, heart failure, 54
 in postural hypotension, 119
- Dextrose, hypertonic See Glucose.
- Diabetes mellitus, coronary disease, 89
 coronary insufficiency, 89
 postural hypotension, 119

- Diamox, 71
 carbonic anhydrase inhibitor, 71
- Diarrhea, coronary insufficiency in, 65
 digitalis, 25
 Mechohyl, 40
 quinidine, 17
- Dibenzamine hydrochloride, 47
 in hypertensive encephalopathy, 133
 in pheochromocytoma, 138
- Dibenzylinc, 47
 in hypertensive encephalopathy, 133
 in peripheral embolism, intra-arterial, 109
 postural hypotension, 119
- Dicumarol, dangers, 106
 hemorrhage, 107
 in coronary insufficiency, 85
 in coronary occlusion, 105
 in heart failure, 64
 in peripheral embolism, 108
 in pulmonary infarction, 101-105
 prothrombin time, 105, 106
 urinalysis, 106
- Dicurin, 66
- Digitalis, 22-29
 atropine, 28
 A-V block, 25, 28
 colored vision, 25
 electrocardiogram, 25
 in arrhythmias, 25, 97
 in atrial fibrillation, 14, 16, 19, 22, 29, 31
 in atrial flutter, 32, 33
 in atrial tachycardia, 38
 in cardiac asthma, 58
 in coronary occlusion, 96
 in elderly people, 61
 in heart failure, 22, 56, 90
 in hyperthyroidism, 29, 30
 in pulmonary edema, 57
 in rheumatic carditis, 29, 30, 124, 125

- Digitalis, in rheumatic heart disease, 29, 31
 in supraventricular tachycardia, 28
 in surgical emergencies, 148
 intravenous, 22, 23
 nausea and vomiting, 25
 potassium, 28
 premature beats, 25, 27, 43, 70
 preparations and properties, Table, 26
 prophylactic, 42
 syncope, 117
 test for underdigitalization, 29
 therapeutic and toxic doses, 24
 toxicity, 25, Figure, 27, 70
 ventricular fibrillation, 47
 ventricular tachycardia, 28, 42
 Digitalis leaf, 24, 26
 Digitoxin, 23, 26
 Digoxin, in arrhythmias, 23-26
 in auricular fibrillation, 23
 in congestive failure, 64
 in emergency kit, 10
 in pulmonary edema, 61
 in ventricular tachycardia, 45
 Dilaudid, in coronary insufficiency, 80
 in coronary occlusion, 93
 in emergency kit, 10
 in pulmonary edema, 59
 in status anginosus, 80
 Dilution hyponatremia, chronic, 70
 intractable edema, 70
 Southey Leach tubes, 70
 Dissecting aneurysm, 139-141
 danger of anticoagulants, 141
 differential diagnosis, 140
 angina pectoris, 140
 coronary insufficiency, 140
 coronary occlusion, 139, 140
 pulmonary infarction, 139
 Dissecting aneurysm, electrocardiogram, 140
 hamopericardium, 140
 in coarctation of aorta, 139
 in pregnancy, 139
 mediastinum, widened, 140
 morphine, 141
 paraplegia, 140
 pleural effusion, 140
 shock, 140
 subacute, 139
 syncope, 140
 with aortic diastolic murmur, 140
 with hypertension, 140
 Distention, atrial tachycardia, 35
 in coronary occlusion, 73
 in digitalis toxicity, 25
 in heart failure, 73
 morphine, 92
 Diuretics, and azotemia, 68
 and electrolyte disturbances, 68
 and hypochloremic alkalosis, 69
 and retention, acute, 70
 and salt depletion, 68, 69
 blood chloride, 68, 69
 blood sodium, 69
 blood urea, 68
 cation exchange resins, effect, 71
 Diamox, 71
 digitalis, toxic effects, 70
 drug effect, 70
 in cardiac asthma, 58
 in coronary occlusion, 90
 in heart failure, 58, 61, 66
 in pulmonary edema, 61, 88
 mercurhydram, 66, 70, 88
 muscle cramps, 67
 Neohydrol, 67
 oral, 67, 69, 71
 prostate, effect, 70
 retention, acute, 70
 salt depletion, 68, 69
 salt restriction, 57, 61, 67
 sodium level, 68, 69

Diuretics, *Thumerm*, 66, 70
uremia, 70

Dizziness *See* Vertigo

Doctor's bag, 9

essential drugs, 9

Dramamine, in congestive failure
74

■ hypertensive encephalopathy, 136

in postural hypotension, 74

Dyspnea, in congestive failure,
57, 96

in pulmonary edema, 58

in pulmonary embolism, 104

paroxysmal nocturnal, 57, 58

E

ECCIMOSIS *See* Purpura

Edema, in heart failure, 54

pulmonary, 58-62

Effort, angina pectoris, 77

and pulmonary edema, 58

Elastic bandages, in postural hypotension, 119

Electric shock, 47

Electrocardiogram, in acute cor pulmonale (pulmonary embolism), 104

in coronary insufficiency, 84

in coronary occlusion, 84, 89-91

in dissecting aneurysm, 140

in pericarditis, acute, 126

in pleochromocytoma, 137

in rheumatic fever, 126

in traumatic heart disease, 144

with digitalis, 25

with potassium toxicity, 28

with quinidine, 21

Electroencephalogram, syncope,
120

Electrolytes, and cation exchange resins, 71

disturbances in congestive failure, 68, 69, 71

in dilution hyponatremia, chronic, 70

Electrolytes, in hypochloremic acidosis, 69

in hyperchloremic alkalosis, 69

in low sodium syndrome, 69

serum determination, 72

Embolectomy, 109

saddle emboli of aorta, 110

Embolism, aminophylline, 109

anticoagulant therapy, 108

arterial, 108

atrial fibrillation, 19

caffeine sodium benzoate, 109

cerebral, 108, 111, 127

Dibenzylamine, 109

embolectomy, 108, 109, 127

femoral pulse, 108

gangrene, 109

hexamethonium, 109

methonium derivatives, 109

peripheral, 91, 108, 109, 111

127

Priscoline, 109

pulmonary, 101-103

saddle embolus, 110

Embryocardia, coronary occlusion, 89

Emergency kit, 10

Emotional disturbances, and coronary insufficiency, 83

and paroxysmal tachycardia, 12, 13

and pulmonary edema, 54

and sinus tachycardia, 51

and supraventricular tachycardia, 33

and syncope, 113

Enemas, in congestive failure, 74

Ephedrine, antidiuretic effect, 70

in A-V block, 50

in Stokes-Adams syndrome, 50

in surgical emergencies, 149

in syncope, 117

Epinephrine, antidiuretic effect,
70

effect on carotid sinus, 36

in A-V block, 50

in coronary occlusion, 49

in emergency kit, 10

- Epinephrine, in postural hypotension, 119
 in Stokes-Adams syndrome, 50
 in surgical emergencies, 152, 153
 in syncope, 115
 pheochromocytoma, 136, 137
 Esophageal leads, atrial flutter, 32
 Esophageal spasm, angina pectoris, 76
 Etamon See Tetraethyl ammonium chloride
 Ether, for hiccups, 75
 surgery, 149
 Ethyl chloride spray, hiccups, 74
 Extrasystoles, 51 See Premature beats
 Eyeball pressure, effect on A-V conduction, 36
 in supraventricular tachycardia, 36

F

- Fainting See Syncope
 Fever, in coronary insufficiency, 84
 in coronary occlusion, 91
 in pulmonary embolism, 104
 Fluids, intake in heart failure, 65
 intravenous, in surgical emergencies, 148
 restriction, 66
 retention, 57
 Flutter waves, 31
 Foci of infection, surgical emergencies, 147
 Fluids restriction in congestive failure, 66
 Friction rub, in coronary occlusion, 91
 in rheumatic fever, 126
 Fruit juices, 65
 in rheumatic fever, 124

G

- GALL BLADDER disease, see angina pectoris, 76
 effect of operation on angina, 148
 Gallop rhythm, in coronary occlusion, 89
 in rheumatic fever, 123
 Gangrene, ice packs, 109
 Gastro-enteritis, and coronary insufficiency, 85
 Ginger ale, for nausea in congestive failure, 65
 Gitalin, 23, 24, 26
 in congestive heart failure, 64
 Glucose, and syncope, 120
 in hypertensive encephalopathy, 132
 Glucose tolerance test, syncope, 121
 Glycosuria, in pheochromocytoma, 136
 Grand mal, in syncope, 115
 Graves' disease See Hyperthyroidism

H

- HEADACHE, in hypertension, 132
 with nitroglycerin, 77
 Heart block See A-V block
 Heart failure, 64-75
 abdominal distention, 73
 ACTH, 124
 and anticoagulants, 64
 and coronary insufficiency, 65
 anemia of bone marrow, 54
 antidiuretic hormone, 53
 drugs, 70
 arrhythmias, 11
 arterio-venous fistula, 55, 57, 72
 ascites, 73
 azotemia, 68
 blood electrolytes, 68-71

- Heart failure, blood, urea, ■■■
 volume, 54
 bloodless phlebotomy, 61
 bronchospasm, 57
 cardiac asthma, 57
 cirrhosis, 73
 output, high, 55
 cervical veins, distention, 57
 chloride level, 69
 colitis, 74
 constipation, 73
 cortisone, 124
 dehydration, 68
 desoxycorticosterone, 54
 dyspnea, 57
 edema, 54
 enemas, 74
 hepatomegaly, 57
 hiccup, 73, 74
 hydrothorax, 72
 hyperchloremic acidosis, 68,
 ■■9
 hypertensive heart disease,
 56
 hypochloremic alkalosis, 69
 hypopotassemia, 69
 in anemia, 56, 72
 in atrial fibrillation, 22
 in beri-beri, 55
 in coronary occlusion, 73, 94
 95, 96, 111
 in cor pulmonale, 62
 in hyperthyroidism, 72
 in Paget's disease, 55
 in pulmonary disease, 62-64
 acute, 62, 63
 chronic, 62
 infarction, 101
 infection, 62, 64
 in rheumatic fever, 124
 in rheumatic heart disease,
 55
 in surgical emergencies, 147,
 148
 in tachycardia, 12
 in traumatic heart disease,
 142
- Heart failure, blood, urea, left
 ventricular failure, 57, 58
 liver disease, 73
 mechanism (pathogenesis),
 54
 "backward" failure, 54,
 55
 cardiac output, 55
 "forward" failure, 54, 55
 nausea, 73, 74
 obesity, 66
 orthopnea, 57
 prostatic disease and diure-
 sis, 70
 pulmonary congestion, 58
 60
 "refractory", 72
 renal blood flow, 56
 right ventricular failure
 acute, 62
 shock, 50, 127
 stroke volume, 55
 sympathetic nerve discharge,
 58
 therapy in elderly people, 70
 treatment, aminophylline, 58,
 68
 ammonium chloride, 68,
 69
 bed rest, 64, 67
 cation exchange resins, 71
 chair treatment, 58
 Diamox, 71
 Dicumarol, 64
 digitalis, 22, 50, 58, 96,
 124
 digitalis toxicity, 70
 potassium loss, 70
 diuretics, 64, 67, 68, 70
 Camestatin, 67
 drug effect, 70
 Mersulhydrin, 68, 70
 mercurial, 64, 70
 Neohydrin, 67
 Dramamine, 71
 fluid intake, 65
 gitalin, 64
 hexamethonium, 62

- Heart failure, treatment, "tonalac", 84
 laxative, 73
 ligation of inferior vena cava, 73
 morphine, 58
 opiates, danger, 73
 Pantopon, 59
 paracentesis, 72
 penicillin, 64
 phlebotomy, 55, 56
 potassium, 89, 124
 protein intake, 73
 radioactive iodine, 73
 sodium, diet, low, 84
 excretion level, 89
 retention, 58, 67
 Thioamerin, 66, 70
 thoracentesis, 72
 Thorazine, 74
 tourniquets, 61
 valvulotomy, 128
 vitamins, 73
 weight reduction, 66
 uremia, 70
 vomiting, 73, 74
 wheezing, 59
 with electrolyte disturbances
 68, 69, 70
 with salt depletion, 68
 poor diet, 73
 restriction, 53, 61, 65
 substitutes, 65
- Heart wound, 142 See Traumatic heart disease
- Hematocrit, acute cardio-pulmonary insufficiency, 63
 pulmonary edema, 61
- Hematuria, quinidine, 21
- Hemiplegia, carotid sinus pressure, 36
- Hemoglobin, 61, 63
- Hemopericardium, due to anticoagulants, 107
 due to dissecting aneurysm, 140
 in acute pericarditis, 92, 107
 in traumatic heart disease, 142, 145
- Hemoptysis, in coronary occlusion, 104
 in mitral stenosis, 128
 in pulmonary embolism, 104
 valvulotomy, 128
- Hemorrhage, gastro-intestinal, 88
 in traumatic heart disease, 142
 pericardial, 107
 subintimal, in coronary occlusion, 89
 surgical emergency, 153
 transfusion, for, 153
 with Dicumarol, 107
 with syncope, 113
- Heparin, clotting time, 105
 in angina pectoris, 78
 in coronary occlusion, 105
 in pulmonary infarction, 105
 protamine sulfate, 105
- Hepatic disease, in congestive failure, 73
 with syncope, 120
- Hepatomegaly, in heart failure, 57
- Herpes zoster, vs angina pectoris, 76
- Hexamethonium, azotemia, danger, 134
 in coronary occlusion, 109
 in heart failure, 62
 in hypertension, 133
 in hypertensive encephalopathy, 133
 in peripheral edema, intra-arterial, 109
 with hypotension, 134
 Trendelenburg position, 134
 neosynephrine, 134
- Hiatus hernia, vs angina pectoris, 76
- Hiccups, in coronary occlusion, 112
 in heart failure, 73
 treatment, 74
 amyl nitrite, 74
 carbon dioxide inhalation, 74
 ether, 75
 ethyl chloride, 74

- Heart failure, blood, urea, 68
 volume, 54
 bloodless phlebotomy, 61
 bronchospasm, 57
 cardiac asthma, 57
 urticaria, 73
 output, high, 55
 cervical veins, distention, 57
 chloride level, 69
 colitis, 74
 constipation, 73
 cortisone, 124
 dehydration, 68
 desoxycorticosterone, 54
 dyspnea, 57
 edema, 54
 enemas, 74
 hepatomegaly, 57
 hiccup, 73, 74
 hydrothorax, 72
 hyperchloremic acidosis, 68,
 69
 hypertensive heart disease,
 56
 hypochloremic alkalosis, 69
 hypopotassemia, 69
 in anemia, 56, 72
 in atrial fibrillation, 22
 in beri-beri, 55
 in coronary occlusion, 73, 94,
 95, 96, 111
 in cor pulmonale, 62
 in hyperthyroidism, 72
 in Paget's disease, 55
 in pulmonary disease, 62-64
 acute, 62, 63
 chronic, 62
 infarction, 101
 infection, 62, 64
 in rheumatic fever, 124
 in rheumatic heart disease,
 55
 in surgical emergencies, 147,
 148
 in tachycardia, 12
 in traumatic heart disease,
 142
- Heart failure, blood, urea, left
 ventricular failure, 57, 58
 liver disease, 73
 mechanism (pathogenesis),
 54
 "backward" failure, 54,
 55
 cardiac output, 55
 "forward" failure, 54, 55
 nausea, 73, 74
 obesity, 66
 orthopnea, 57
 prostatic disease and diure-
 sis, 70
 pulmonary congestion, 58,
 60
 "refractory", 72
 renal blood flow, 56
 right ventricular failure,
 acute, 62
 shock, 59, 127
 stroke volume, 55
 sympathetic nerve discharge,
 58
 therapy in elderly people, 70
 treatment, aminophylline, 58,
 68
 ammonium chloride, 68,
 69
 bed rest, 64, 67
 cation exchange resins, 71
 chair treatment, 58
 Diamox, 71
 Dicumarol, 64
 digitalis, 22, 56, 58, 90,
 124
 digitalis toxicity, 70
 potassium loss, 70
 diuretics, 64, 67, 68, 70
 Cumertalin, 67
 drug effect, 70
 Mercurhydrin, 66, 70
 mercurial, 64, 70
 Neohydrin, 67
 Dramamine, 74
 fluid intake, 65
 gitaba, 64
 hexamethonium, 62

- Heart failure, treatment, "tonalac", 54
 laxative, 73
 ligation of inferior vena cava, 73
 morphine, 58
 opiates, danger, 73
 Pantopon, 59
 paracentesis, 72
 penicillin, 64
 phlebotomy, 55, 56
 potassium, 69, 124
 protein intake, 73
 radioactive iodine, 73
 sodium, diet, low, 64
 excretion level, 69
 retention, 56, 67
 Thomerin, 66, 70
 thoracentesis, 72
 Thorazine, 74
 tourniquets, 61
 valvulotomy, 128
 vitamins, 73
 weight reduction, 66
 urcemia, 70
 vomiting, 73, 74
 wheezing, 59
 with electrolyte disturbances, 68, 69, 70
 with salt depletion, 68
 poor diet, 73
 restriction, 55, 64, 65
 substitutes, 65
- Heart wound, 142. See Traumatic heart disease
- Hematocrit, acute cardio-pulmonary insufficiency, 63
 pulmonary edema, 61
- Hematuria, quinidine, 21
- Hemiplegia, carotid sinus pressure, 36
- Hemoglobin, 61, 63
- Hemopericardium, due to anticoagulants, 107
 due to dissecting aneurysm, 140
 in acute pericarditis, 92, 107
 in traumatic heart disease, 142, 145
- Hemoptysis, in coronary occlusion, 104
 in mitral stenosis, 128
 in pulmonary embolism, 104
 valvulotomy, 128
- Hemorrhage, gastro-intestinal, 88
 in traumatic heart disease, 112
 pericardial, 107
 subintimal, in coronary occlusion, 89
 surgical emergency, 153
 transfusion, for, 153
 with Dicumarol, 107
 with syncope, 113
- Heparin, clotting time, 105
 in angina pectoris, 79
 in coronary occlusion, 105
 in pulmonary infarction, 105
 protamine sulfate, 105
- Hepatic disease, in congestive failure, 73
 with syncope, 120
- Hepatomegaly, in heart failure, 57
- Herpes zoster, vs angina pectoris, 76
- Hexamethonium, azotemia, danger, 134
 in coronary occlusion, 109
 in heart failure, 62
 in hypertension, 133
 in hypertensive encephalopathy, 133
 in peripheral embolism, intra-arterial, 109
 with hypotension, 134
 Trendelenburg position, 134
 neosynephrine, 134
- Hiatus hernia, vs angina pectoris, 76
- Hiccup, in coronary occlusion, 112
 in heart failure, 73
 treatment, 74
 amyl nitrite, 74
 carbon dioxide inhalation, 74
 ether, 75
 ethyl chloride, 74,

- Hiccup, treatment, mazin, 74
 phrenicectomy, 75
 quinidine, 75
 sedation, 74
 Thorazine, 75
 Histamine, test for pheochromocytoma, 137
 Homan's sign, in pulmonary embolism, 104
 in venous thrombosis, 104
 Hospital vs home care, in coronary occlusion, 94
 Hunger, syncope, 120
 Hydergine, in angina pectoris, 79
 Hydrothorax, in heart failure, 72
 Hykinone See Vitamin K
 Hyperchloremic acidosis, 69, 70
 with ammonium chloride, 69
 with sodium bicarbonate for, 70
 Hyperinsulinism, pancreatic adenomata, 120
 syncope, 120
 Hypertension, encephalopathy, 136
 hexamethonium, 62, 133
 in dissecting aneurysm, 140
 in pheochromocytoma, 136
 malignant, 132
 Hypertensive crises See Hypertensive encephalopathy
 Hypertensive encephalopathy, 132-136
 in nephritis, chronic, 132
 in pheochromocytoma, 136
 symptoms, coma 132
 convulsions, 132
 headache, 132
 nausea, 136
 paralysis, 132
 vomiting, 136
 treatment, Ansolysen, 135
 anti-hypertensive agents, 132
 Apresoline, 133, 135
 Arfonad, 135
 Hypertensive encephalopathy, treatment, dextrose, 132
 Dibenamine, 133
 Dibenzyline, 133
 Dramamine, 136
 hexamethonium, 133
 magnesium sulfate, 132
 Marazine, 136
 spinal tap, 132
 Thorazine, 136
 Verloind, 133
 with coronary insufficiency, 85
 with uricemia, 132
 Hypertensive heart disease, an esthesia, in, 149
 with cardiac failure, 56
 Hyperthyroidism, and angina pectoris, 82
 digitalis, 29
 with atrial fibrillation, 13, 14, 18, 29
 with atrial tachycardia, 13
 with congestive failure, 72
 with high cardiac output, 55
 Hyperthyroidism, Lugol's solution, 30
 Mecohyl, 40
 propyl thiouracil, 30
 protein-bound iodine, 13
 quinidine, 30
 radioactive iodine, 118, 13, 30
 supraventricular tachycardia, 35
 Hyperventilation, and postural hypotension, 118, 119
 and syncope, 119, 120
 Hypochloremic alkalosis, 69
 ammonium chloride, 69
 carbon dioxide combining power, 69
 hydrochloric acid, 69
 Hypoglycemia, and coronary insufficiency, 69
 and syncope, 120
 convulsions, 120
 glucose, 120

- Hypopotassemia, cation exchange resins, and 71
 in congestive failure, 69
 Hypotension. *See* Postural hypotension
 and coronary insufficiency, 85
 in surgical emergencies, 149
 Hypothermia, cardiac surgery, 131
 Hypothyroidism, induced for angina pectoris, 81-83
 Hysteria, syncope, 119

I

- I₁₃₁*. *See* Radioactive iodine
 Ice packs, in gangrene, 109
 Idioventricular pacemaker, Stokes-Adams syndrome, 49
 Infarction, angina pectoris, 76
 myocardial, 89
 pulmonary, 104
 traumatic heart disease, 144
 Infections, and coronary insufficiency, 85
 and supraventricular tachycardia, 35
 Intratracheal tube, in cardiac arrest, 153
 Ipecac, syrup of in supraventricular tachycardia, 39
 Ischemia, myocardial, in coronary insufficiency, 84
 Isuprel, in A-V block (Stokes-Adams), 50

J

- Joints, effusions, in rheumatic fever, 113
 pains, in rheumatic fever, 113
 salicylates, 113, 126

K

- KHELLIN, in angina pectoris, 79
 nausea and vomiting, 79

L

- LANATOSIDE C. *See* Cechlanid
 "Lonalac" powder, 64
 Laxatives, cascara sagrada, 74
 in heart failure, 73
 milk of magnesia, 74
 Lethargy, in salt depletion, 68
 Leucocytosis, in coronary insufficiency, 86
 in coronary occlusion, 105
 in pulmonary infarction, 105
 Levophed, in coronary occlusion, 94, 95
 in shock, 138
 in surgical emergencies, 149
 Ligation of inferior vena cava for congestive heart failure, 73
 Low sodium syndrome, 69
 digitalis toxicity, 69
 hypertonic sodium chloride, 69
 potassium deficit, 69
 potassium therapy, 69
 Liver disease, in congestive failure, 73
 Lugol's solution, 30
 Magnesium sulfate, in hypertensive encephalopathy, 132
 in ventricular fibrillation, 49
 in ventricular tachycardia, 46
 intravenous, 46
 nausea, 46
 vertigo, 46
 weakness, 46
 Marazine, in hypertensive encephalopathy, 136
 Meils, arrhythmias, 13
 and pulmonary edema, 58
 small, 65
 Methylol, 40
 allergy, 40
 and asthma, 40
 atropine, 38, 40
 carotid sinus effect, 36
 in atrial tachycardia, 36, 40
 reactions, 40

- Mecholyl, with atrial fibrillation, 40
 Mediastinum, widened, in dissecting aneurysm, 140
 Mercuhydrin. *See* Diuretics
 Mercurial diuretics. *See* Diuretics
 Metamine, in angina pectoris, 79
 Metapon, in coronary occlusion, 93
 Methionum derivatives (C₆, C₈)
 See Hexamethonium
 Milk of magnesia, in congestive failure, 74
 Milk, powdered, "Lonalac", 64
 Mitral stenosis, 126-131
 anticoagulant therapy, 127
 atrial fibrillation, 126
 atrial thrombi, 126
 ball valve thrombus, 126
 cyanosis, 126
 embolectomy, 127
 hemoptysis, 128
 left auricular appendectomy, 127
 operations on mitral valve, 128
 peripheral embolism, 127
 pulmonary edema, 128
 Mitral valvuloplasty. *See* Mitral valvulotomy
 Mitral valvulotomy, cardiac arrest, 130
 contraindications, acute rheumatic fever, 129
 left ventricular enlargement, 129
 subacute bacterial endocarditis, 129
 indications, congestive failure, 128
 hemoptysis, 128
 mitral stenosis, 128
 pulmonary edema, 128
 right ventricular enlargement, 128
 "post-commissurotomy syndrome", 130
 Morphine, antidiuretic effect, 70
 in acute pericarditis, 126
 in coronary occlusion, 92, 93
 in dissecting aneurysm, 141
 in emergency kit, 10
 in heart failure, 58
 in pulmonary edema, 58, 59
 in traumatic heart disease, 143
 in ventricular tachycardia, 46
 intravenous, 46, 92
 Muscle cramps due to diuresis, 67
 Myocardial infarction. *See* Coronary occlusion
 Myocardium, contusion, 145, 146
 infarction. *See* Coronary occlusion
 laceration, 143
- N
- NAUSEA, from Kiehl, 79
 from Mecholyl, 36
 from morphine, 73
 from Pronestyl, 45
 from quainidine, 20
 in congestive failure, 74
 in coronary occlusion, 112
 in hypertensive encephalopathy, 136
 in pheochromocytoma, 136
 syncope, 113
 treatment, 74
 Dranamine, 74, 136
 Marcaine, 136
 Thorazine, 74, 136
 with digitalis, 25
 Neohydrin, 67
 Neostigmine, in atrial tachycardia, 40
 Neosynephrine, carotid sinus, 36
 following use of hexamethonium, 134
 in cardiac standstill, 45
 in coronary occlusion, 93
 in emergency kit, 10
 in postural hypotension, 119

- Neosynephrine, in supraventricular tachycardia, 36, 39
 in surgical emergencies, 89, 149
 syncope, 115, 118
 ventricular premature beats, danger, 39
- Nephritis, acute, 132
 and hypertensive encephalopathy, 132
- Neurocirculatory asthenia, and postural hypotension, 118
- Niacin, intravenous, for hiccup, in congestive failure, 74
- Nicotinic acid, in angina pectoris, 79
- Nitrites, ineffective in coronary occlusion, 76
- Nitroglycerin, 77-78
 flushes, 77
 headache, 77
 in angina pectoris, 77
 in coronary occlusion, danger, 93
 in emergency kit, 10
 prophylactic, 77
 syncope, 77, 119
 throbbing, 77
- Nitroglyn, in angina pectoris, 79
- Nodal tachycardia, Figure, 37
 See Atrial tachycardia
- Nor-epinephrine (Levophed), in coronary occlusion, 95
 in pheochromocytoma, 136, 137
- O
- Obesity, and heart failure, 66
 and surgical emergencies, 150
- Occlusion, arterial cerebral, 108
 chronic Dicumarol therapy, 127
 in dissecting aneurysm, 140
 in rheumatic heart disease, 127
- Orthopnea, heart failure, 57
- Ouabain See Strophanthus G
- Oxygen therapy, for hiccup, 75
 in congestive failure, 59, 63
- Oxygen therapy, in coronary occlusion, 93, 95
 in pulmonary edema, 59, 60
 in surgical emergencies, 148
 under increased pressure, 59
- P
- PACEMAKER, A-V block, 51
- Paget's disease, 55
- Palpitation, 12
 in pheochromocytoma, 136
- Pancarditis, 123
- Pancreatitis, vs coronary insufficiency, 85
- Pantopon, in coronary occlusion, 93
 in pulmonary edema, 59
- Papaverine, in angina pectoris, 79
- Paracentesis abdominis, in congestive failure, 72
- Paraldehyde, in acute cardiopulmonary insufficiency, 63
- Paralysis, in hypertensive crises, 132
- Paraplegia, in dissecting aneurysm, 140
- Parasympathomimetic drugs, acetylcholine, 40
 atropine, 36, 40
 in supraventricular tachycardia, 40
 Mecholyl, 36, 40
 neostigmine, 40
- Paravertebral block, alcohol, for angina pectoris, 82
- Paredrine, in postural hypotension, 119
 in Stokes-Adams syndrome, 50
 in syncope, 118
- Paroxysmal atrial fibrillation See Atrial fibrillation.
- Paroxysmal tachycardia, 11, 12, Tables, 15, 41 See Atrial fibrillation, Atrial flutter, Atrial tachycardia

- Penicillin, in heart failure, 64
 Pepper, 65
 Peptic ulcer vs angina pectoris, 76
 Pericardial effusion, in dissecting aneurysm, 140
 in rheumatic fever, 126
 in traumatic heart disease, 142
 salicylates, for, 126
 Pericardial rub, in rheumatic fever, 126
 in traumatic heart disease, 126
 traumatic heart disease, 142
 Pericarditis, acute, anticoagulants, danger, 107
 electrocardiogram, Figure, 125, 126
 friction rub, 126
 in coronary occlusion, 91, 92, 107
 in rheumatic fever, 124, 126
 in traumatic heart disease, 144
 morphine, 126
 Peripheral resistance, and syncope, 113
 Perisrate, in angina pectoris, 78
 Pernicious anemia, and heart failure, 72
 Perspiration *See* Sweating
 Pheochromocytoma, 136-138
 and postural hypotension, 118
 basal metabolic rate, in, 136
 electrocardiogram, 137
 epinephrine, 136, 137
 nor-epinephrine, 136, 137
 symptoms, : abdominal pain, 136
 glycosuria, 136
 nausea, 136
 palpitation, 136
 sweating, 136
 tachycardia, 136
 vertigo, 136
 vomiting, 136
 tests, Benzodioxane, 137
 Pheochromocytoma, tests, histamine, 137
 Regitine, 137
 treatment, Dibenzamine, 138
 Dibenzylinc, 138
 Levophed, 138
 surgery, 138
 with hypertensive encephalopathy, 136
 Phlebotomy, acute cardio-pulmonary insufficiency, 63
 bloodless (tourniquets), 61
 in heart failure, 55, 56, 57
 in pulmonary edema, 61
 needles, emergency kit, 10
 Phrenicectomy, for hiccups, 75
 Plasma, in traumatic heart disease, 143
 Pleural effusion, in dissecting aneurysm, 140
 in heart failure, 72
 Pleurisy vs angina pectoris, 76
 Pneumonia, and auricular fibrillation, 14
 Pneumothorax vs angina pectoris, 76
 Polomyelitis, and pulmonary edema, 58
 "Post-commissurotomy syndrome", 130
 Postural hypotension, 118-119
 cardiac output, 118
 etiology, Addison's disease, 118
 amyl nitrite, 119
 cord tumor, 118
 diabetes mellitus, 118
 Dibenzylinc, 119
 hexamethonium, 134
 hyperventilation, 118
 hysteria, 119
 neurocirculatory asthenia, 118
 nitroglycerin, 119
 pheochromocytoma, 118

- Postural hypotension, etiology, sympathectomy, 118
 tabes dorsalis, 118
 Thorazine, 74
 syncope, 118
 treatment, abdominal belt, 110
 atropine, 119
 bed, tilted, 119
 desoxycorticosterone, 119
 elastic bandages, 119
 epinephrine, 119
 neosynephrine, 119
 Paredrine, 119
 salt, 119
- Potassium-merum, ACTH, effect, 124
 congestive failure, 69, 124
 depletion, 28
 determination, 69
 digitalis toxicity, 28
 diuretics, effect, 70
 electrocardiogram, effect, 28
 toxicity, 28
- Potassium salts, in arrhythmia, 28
 in atrial fibrillation, 22, 28
 in digitalis toxicity, 28
 in emergency kit, 10
 in fruit juices, 28, 124
 in low sodium syndrome, 69
 in ventricular fibrillation, 49
 in ventricular tachycardia, 46
 toxic effects, 28
 "Triplex", 22, 28
- Premature beats, 51-53
 and paroxysmal tachycardia, 53
 anxiety, 52
 arrhythmia, 52
 digitalis, 23, 43
 in congestive failure, 50
 in coronary occlusion, 53, 97, 99
 in surgical procedures, 51
 neosynephrine, 59
 treatment, alcoholic beverages, elimination, 53
- Premature beats, treatment, Pro-
 nestyl, 52
 psychiatric therapy, 52
 quinidine, 52
 sedation, 52
 smoking, elimination, 52
 ventricular, 51-53
- Precipitous phase, of coronary
 occlusion, 85, 89
- P-R interval, prolonged, in cor-
 onary occlusion, 99
 in digitalis, 23
- Priscoline, in peripheral embol-
 ism, intra-arterial, 109
- Proanthracol for syncope, 118
- Procaine amide See Pronestyl
- Procaine, intravenous, in surgical
 procedures, 46, 49
- Pronestyl (procaine amide), a-
 granulocytosis, 45
 and purpura, 45
 coronary occlusion, 97
 electrocardiogram, 43
 in atrial fibrillation, 22
 in atrial flutter, Table, 41
 in atrial tachycardia, 38, 39
 in cardiac catheterization, 43
 standstill, 132
 in emergency kit, 10
 in surgical procedures, 43
 149, 150, 153
 intravenous, 45
 in ventricular fibrillation, 47
 152
 premature beats, 53
 prophylactic, 42, 46
 tachycardia, 43-46, Fig-
 ure, 44, 97
- Propral thioracil, in angina de-
 cubitus, 81
 in arrhythmia, 30
 in hyperthyroidism, 30
- Prostatic disease, indwelling cath-
 eter, 70
 mercaptals, effect, 70
- Protamine sulfate, hypotension,
 105
- Protein intake, in congestive fail-
 ure, 73

- Prothrombin time, in Dicumarol therapy, 103
- Psychiatric therapy, in premature beats, 52
in syncope, 120
- Pulmonary congestion, aminophylline, 60
in coronary occlusion, 58, 89, 96
in heart failure, 58, 60
mercurial diuretic, 61, 68
morphine, 58, 59
oxygen, 59, 60
- Pulmonary disease, chronic, and cardiac failure, 53, 62
- Pulmonary edema, acute, 58-62
bubbling rales, 59
cardiac output, 53
Cheyne-Stokes respiration, 60
cyanosis, 58
dyspnea, 58
emotional disturbances, 58
exercise, 58
hematocrit, 61
in coronary occlusion, 58, 89, 96
in mitral stenosis, 128
in surgical emergencies, 148
nitals, large, 58
paroxysmal tachycardia, 58
pneumonia, 58
shock, 60, 61
trauma, 58
treatment, alcohol vapor, 62
aminophylline, 60
anti-foaming agents, 62
atropine, 59
Cedidid, 60
Demerol, 59
digoxin, 61
Dilaudid, 59
diuretics, mercurial, 61, 88
hexamethonium, 62
mitral valvulotomy, 128
morphine, 59
ouabain, 60
- Pulmonary edema, treatment, oxygen, 59, 60
packed red blood cells, 62
Pantopon, 59
phlebotomy, 61
bloodless, 61
strophanthin, 60, 61
venous pressure, 61
wheezing, 59
- Pulmonary embolism, 101-103
acute cor pulmonale, 62-63
anticoagulant therapy, 101, 104
cough, 104
differential diagnosis, 102, 104
dyspnea, 104
electrocardiogram, Figure, 103, 104
fever, 104
hemoptysis, 104
Homan's sign, 104
in coronary occlusion, Table, 102
right bundle branch block, 104
shock, 104
tachycardia, 104
with coronary insufficiency, 104
- Pulmonary infarction, acute cor pulmonale, 64
anticoagulant therapy, 101-103
fever, 104
in congestive failure, 101
in rheumatic heart disease, 127
in surgical emergencies, 150
leucocytosis, 103
- Pulmonary infection, acute cor pulmonale, 64
in congestive failure, 62, 61
penicillin, for, 63
- Pulse deficit, in auricular fibrillation, 14
- Purpura, from Pronovyl, 15
from quinine, 20

Q

QUINIDINE, 16-22

- antidiuretic effect, 70
- antihistamines, 21
- carotid sinus, 36
- concoals of, 18
- idiosyncrasy, 20
- in atrial fibrillation, 14-19, 30
- in atrial flutter, 32, 33
- in atrial tachycardia, 36, 38
- in A-V block, 21
- in bundle branch block, 21
- in coronary occlusion, 97
- in emergency kit, 10
- in hiccup, 75
- in hyperthyroidism, 18, 30
- "in oil", 18
- in premature beats, 52
- in supraventricular tachycardia, 21, 36, 38
- in surgical emergencies, 149
- in ventricular fibrillation, -47, 49
- in ventricular tachycardia, 43, 97, 98
- intramuscular, 17
- intravenous, 17
- maintenance dose, 18, 33
- Necholyl, 36
- plasma concentration, 17
- prophylactic 18
- "Quinidate", 18
- rheumatic fever, 18, 31
- sensitivity, 16, 20
- sinus rhythm, restoration 19
- toxic effects, 17
- "cinchonism", 20
- electrocardiogram, 21
- hematuria, 21
- laryngeal edema, 20
- purpura, 20, 21

R

RADIOACTIVE iodine in angina pectoris, 81-82

Radioactive iodine in congestive heart failure, 73

in hyperthyroidism 30

Regitine, -47

in pheochromocytoma 137

Renal function, blood flow, 56
excretion, 56

Reserpine, in angina pectoris 74, 60

Respiration, artificial during operation, 151, 153

Cheyne-Stokes aminophylline, 60

quinidine depression, 20

Respirators in acute cardio-pulmonary insufficiency, 63

Rheumatic fever, 30, 31, 123-126
ACTH 123, 124, 126

adrenal cortical hormones, 123

arrhythmias, 29, 31, 123

atrial fibrillation, 18, 29, 31, 123

blood potassium, 124

cardiac tamponade, 126

carditis, 31, 123

congestive failure, 123, 124, 125

cortisone, 123, 124, 126

digitalis, 29, 31, 124

electrocardiogram, 126

friction rub, 126

fruit juices, 124

gallop rhythm, 123

joint pains, 123

mitral valvulotomy, 129

morphine, 120

pericarditis, 123

pericardial effusion, 121

tapping, 120

pericarditis, acute, 123, 121

Figure, 125, 126

quinidine, 18, 31

salicylates, 123, 126

sedimentation rate, 123

shock, 124

tachycardia, 123

Rheumatic heart disease, 126-131

Rheumatic heart disease, anticoagulants, 127
 ascites, 73, 126
 atrial fibrillation, 126, 129
 atrial thrombi, 126
 bacterial endocarditis, subacute, 129
 ball valve thrombus, 126
 cardiac cirrhosis, 73
 commissurotomy *See* Mitral valvulotomy
 congestive failure, 128
 cyanosis, 126
 Dicumarol, 127
 digitalis, 29, 31, 124
 dyspnea, 126, 128
 embolectomy, 127
 embolization, 127
 hemoptysis, 128
 left auricular appendectomy, 127
 mercurial injections, 125
 mitral insufficiency, 127, 128
 mitral stenosis, 126-128
 mitral valvulotomy, 128
 morphine, 126
 oxygen, 127
 "post-commissurotomy syndrome", 130
 premature beats, 19
 pulmonary edema, 128
 pulmonary infarction, 127
 right ventricular enlargement, 128
 shock, 128
 surgical emergencies, 148
 ventricular tachycardia, 42

Rupture of the heart, 146

S

SADDLE embolism, 110
 Salicylates, in rheumatic fever, 123
 Saline, intravenous, in surgical emergencies, 148

Salivation, with Mecholyl, 40
 Salt, depletion, with diuretics, 111
 in heart failure, 55
 in postural hypotension, 119
 restriction, 55, 64, 65, 96
 Salt substitutes, in heart failure 65

potassium chloride, 65
 Scotomata, in digitalis therapy, 25

Sedatives, for hiccups in congestive failure, 74
 in angina pectoris, 80
 in atrial fibrillation, 14
 in paroxysmal tachycardia, 13
 in syncope, 120

Sedimentation rate in coronary insufficiency, 84
 in coronary occlusion 91
 in rheumatic fever, 123

Sexual intercourse, in coronary insufficiency, 88

Shock, epinephrine, 50
 from Mecholyl, 40
 in arrhythmias, 12
 in A-V block, 50
 in congestive failure 58
 in coronary occlusion, 89, 94 95
 in dissecting aneurysm, 140
 in paroxysmal tachycardia, 12
 in pericarditis, 126
 in pulmonary edema, 58
 in pulmonary embolism, 104
 in traumatic heart disease, 142 143

in ventricular fibrillation, 47
 morphine sulfate, 59
 oxygen, 95
 peripheral, 59
 postoperative, in coronary insufficiency, 88
 Trendelenburg position, 95

Sinus bradycardia, 51

atropine, 52
 carotid sinus stimulation, 51
 digitalis, 25
 dizziness, 51

- Sinus bradycardia, ephedrine, 52
syncope, 51
- Sinus rhythm, restoration with
quinidine, 19
- Sinus tachycardia, differential di-
agnosis, Table, 15
hyperthyroidism, 51
radioactive iodine, 51
- Skin eruptions with quinidine, 20
- Sodium bicarbonate, salt-poor
diet, 65
- Sodium chloride, hypertonic, in
low sodium syndrome, 69
- Sodium, serum level, 69
depletion, 68
diet, low in, 64
effect of exercise, 67
excretion, 56
in congestive failure, 64
in surgical emergencies,
148
retention, 56, 67, 124
- Spinal cord tumor, postural hypo-
tension, 118
- Spinal tap, in hypertensive en-
cephalopathy, 132
- Spondylitis vs angina pectoris,
711
- Stab wound of heart, 142
- Status anginosus, 78, 80
Dilaudid, 80
nitroglycerin, 80
- Steering wheel accidents, 145
- Stokes-Adams syndrome, digi-
tals overdosage, 50
in heart block, 49
syncope, 118
treatment, ephedrine, 50
epinephrine, 50, 90
Isuprel, 50
pacemaker, 50
Paraldrine, 50
thumping on precordium,
50
in ventricular fibrillation, 47
- Stroke volume, in cardiac failure,
55
- Strophanthus (K and G), 26
- Strophanthus (K and G), in
atrial fibrillation, 22
in coronary occlusion, 98
in emergency kit, 10
in pulmonary edema 60
in supraventricular tachy-
cardia, 38
in ventricular tachycardia,
Figure, 44
"Strophosid", 22
- Subacute bacterial endocarditis,
valvulotomy, contraindicated,
129
- Subendocardium, ischemia, in
angina pectoris, 77
in coronary insufficiency, 84
necrosis, 84
- Sudden death, in ventricular
fibrillation, 47
- Supraventricular tachycardia, 12
See Atrial fibrillation,
Atrial flutter, Atrial tachy-
cardia
differential diagnosis, Table,
15
- Surgical emergencies, 147-154
age, 147
ambulation, 150
and angina pectoris, 68, 148
anesthesia, 88, 147, 149
arrhythmias, 147, 149, 150
artificial respiration, 151,
153
atropine, 151
calcium chloride, 152
cardiac arrest, 50, 151-154
compression, 152, 153
resuscitation, 152
cholecystectomy, 148
CO₂, 153
coronary insufficiency, 147,
149
occlusion, 148
cyclopropane, 149
arrhythmias, 149, 150
defibrillation, 152
digitalization, 148
endotracheal tube, 153

- Surgical emergencies, ephedrine, 149
 epinephrine, 152, 154
 ether, 149
 fluid intake, 148
 heart failure, 147, 148
 hemorrhage, 88, 153
 hypotension, 149
 drugs, 149
 Levophed, 149
 mouth to mouth breathing, 153
 myocardial infarction, 148
 neosynephrine, 89, 149
 oxygen, 148, 152
 postoperative thromboembolism, 150
 Procaine, 152
 Pronestyl, 149, 150
 pulmonary edema, 148
 infarction, 150
 quinidine, 149
 rheumatic heart disease, 148
 saline intravenous, 148
 sodium intake, 148
 spinal anesthesia, 149
 Stokes-Adams syndrome, 150
 Thiopental, 149
 thumping on chest, 154
 transfusion, 153
 Vasovyl, 149
 ventricular fibrillation, 149, 150, 152
 tachycardia, 150
- Surgical procedures, anastomosis of aorta to coronary sinus, 83
 auricular appendectomy, 127
 cardiopericardioplexy, 82, 83
 embolectomy, 109
 excision of carotid sinus, 118
 mitral valve, 128-129
 phrenicectomy, 75
 thoracic sympathectomy, 82
 thyroidectomy, 81
 traumatic heart disease, 143
- Sweating in coronary occlusion 89
- Sweating, in pheochromocytoma, 136
 syncope, 115
- Sympathectomy, postural hypotension, 118
 thoracic, in angina pectoris, 82
- Sympathetic nerve block, 82
- Syncope, 113-122
 anxiety, 120
 cardiac output, 113
 carotid sinus, 115, 117, 118
 cerebral anoxia, 113, 122
 arteriosclerosis, 113, 122
 clonic twitching, 115
 convulsions, 118, 120
 diagnosis and treatment, Table, 114
 digitalis, 117
 dizziness, 51
 electroencephalogram, 120
 emotional disturbances, 113
 epinephrine, 115
 fainting, 113
 from fright, 113
 from heating pads, 115
 from nitroglycerin, 77, 119
 from sight of blood, 113
 glucose tolerance test, 121
 grand mal, 115
 heart disease, 121
 hysteria, 119
 in anemia, 122
 in aortic stenosis, 121
 in arrhythmias, 11, 12, 51, 121
 in asystole, 50, 121
 in A-V block, 121
 in bradycardia, 51
 in calcific aortic stenosis, 121
 in coronary occlusion, 121
 in coughing bouts, 113, 120
 in dissecting aneurysm, 140
 in hemorrhage, 113
 in hepatic disease, 120
 in hunger, 120
 in overactive pancreas, 120
 in pancreatic adenomata, 120
 in postural hypotension, 118-119

- Syncope, in sinus bradycardia, 51
 in Stokes-Adams syndrome, 50, 121
 neurogenic, 113
 peripheral resistance decreased, 113
 straining at stool, 120
 suggestion, 120
 tight collars, 117
 treatment, atropine, 118
 Banthine, 118
 denervation, 118
 ephedrine, 118
 glucose, 120
 neosynephrine, 115, 118
 Paradrine, 118
 Pro-Banthine, 118
 psychiatric, 120
 sedation, 120
 Valsalva maneuver, 120
 vasodepressor, 113, Table, 114
 with hyperventilation, 119
 with hypoglycemia, 120
- T**
- Tabs dorsalis, and postural hypotension, 118
 Tachycardia, differentiation, 15
 nodal, 12, Figure, 37
 paroxysmal, 11, 12
 sinus, 51
 supraventricular, 12-15, 28
 ventricular, 42-47
 Tamponade, cardiac, in rheumatic fever, 128
 in traumatic heart disease, 143
 Testosterone, in angina pectoris, 79
 Thiamin, in emergency kit, 10
 in heart failure, 66, 70
 Thiopental, 149
 Thoracentesis, in congestive failure, 72
 Thorazine, in congestive heart failure, 74
 Thorazine, in hiccup, 75
 in hypertensive encephalopathy, 136
 postural hypotension, 74
 Thrombosis, atrial, 126
 ball valve thrombus, 126
 coronary See Coronary occlusion
 in auricular fibrillation, 19
 in mitral stenosis, 126
 mural, in coronary occlusion, 91
 venous, Homan's sign, 104
 Thumping on chest wall, in cardiac arrest, 154
 Thyroidectomy, in angina pectoris, 81
 Thyrotoxicosis See Hyperthyroidism
 Tinnitus, due to quinidine, 20
 Tobacco, in atrial tachycardia, 13, 35
 in coronary insufficiency, 88
 supraventricular tachycardia, 35
 Tourniquets, in bloodless phlebotomy, 61
 in emergency kit, 10
 Transfusion, in anticoagulant therapy, 107
 in coronary disease, 107
 in coronary insufficiency, 88, 89
 in coronary occlusion, 94
 in pulmonary edema, with packed red blood cells, 62
 in surgical emergency, 153
 in traumatic heart disease, 143
 intra-arterial, 94
 Traumatic heart disease, 142-146
 bullet wound, 142, 144
 cardiac arrest, 143
 cardiac tamponade, 142, 143
 congestive heart failure, 142
 coronary insufficiency, 145
 coronary occlusion, 144

- Traumatic heart disease, criteria for cardiac damage, 145-146
 differential diagnosis, 145-146
 electrocardiogram, 144
 hemopericardium, 142, 145
 hemorrhage, 142
 in boxes, 144
 morphine, 143
 myocardial contusions 145, 146
 myocardial laceration, 143
 penetrating wound 142
 pericardial tap, 142
 plasma, 143
 rupture of the heart, 146
 shock, 142
 "steering wheel" accidents, 145
 transfusion, 143
 with acute pericarditis, 144
 with arrhythmias, 145
 with atrial fibrillation, 145
 x-ray, 144
- Trendelenburg position, in coronary occlusion, 95
 in shock, 95
 in use of hexamethonium, 134
- Trigeminal rhythm, digitalis, 25
- Tromexan, 105

U

- UREA, blood, during mercurial diuretic therapy, 68
- Uremia, from mercurial diuretics, 70
 hypertensive encephalopathy, 132
- Urinalysis, in Diaminol therapy, 100
- Urinary retention, mercurials, 70

V

- Vagal reflexes, 38
 stimulation, in supraventricular tachycardia, 35, 39
 stimulation, in surgical emergencies, 151
- Valsalva maneuver, in syncope, 120
- Vasopressor drugs, in coronary occlusion, 94
 Levophed, 94
 neosynephrine, 95
 Vasovyl, 95
 Wyamine, 95
- Vasovyl, in atrial tachycardia, 39
 in coronary occlusion, 95
 in surgical emergencies, 149
- Venous pressure in pulmonary edema, 61
- Venous pulsations, atrial flutter, 31
 in ventricular tachycardia, 42
- Ventricular aneurysm, 111
- Ventricular enlargement, left, valvulotomy as contraindication, 129
- Ventricular enlargement, right, valvulotomy, 128
- Ventricular fibrillation 11, 47-49
 anesthesia, 47
 atropine, 49
 cardiac standstill, 49
 convulsions, 47
 etiology, coronary occlusion, 47, Figure, 48
 digitalis, 47
 electric shock 47
 Stokes-Adams syndrome, 47, Figure, 48
 sudden death, 47
 surgical procedures, 49, 140, 150, 152
 treatment, magnesium sulfate, 49
 oxygen, 49
 potassium, 49

- Ventricular fibrillation, treatment,
 Procaine, 49
 Pronestyl, 47, 49
 quinidine, 47, 49
- Ventricular tachycardia, 11, 42-47
 bundle branch block, 31
 from digitalis, 42
 in cardiac catheterization, 42
 in coronary disease, 13, 42
 in coronary occlusion, 13, 44, 97
 in rheumatic heart disease, 42
 in surgical emergencies, 150
- Mercurhydriol, 45
- prevention, adrenergic blocking agents, 47
 Procaine, 46
- treatment, atabrine, 46
 magnesium sulfate, intravenous, 46
 morphine, intravenous, 46
 potassium chloride, 46
 salts, 46
- Pronestyl, 43-46, Figure, 44, 150
 quinidine, 43
- venous pulsations, 42
- venous pulse tracings, 42
- with A-V block, 45
- with cyclopropine anesthesia, 47
- Wolff-Parkinson-White syndrome, 12
- Venodil, in hypertensive encephalopathy, 133
- Vertigo, in arrhythmias, 11
 in pheochromocytoma, 136
 in sinus bradycardia, 51
 in syncope, 51
- magnesium sulfate, 46
- Visual disturbances, digitalis, 25
 quinidine, 20
- Vitamin E (Alpha Tocopherol), in angina pectoris, 79
- Vitamin K, in Dicumarol therapy, 107
- Vitamin K, oxide, 106
- Vitamins, intake in congestive failure, 73
- Vomiting, digitalis toxicity, 25
- Dramamine, 74
- for tachycardias, induction, 39
- in arrhythmias, 11
- in congestive failure, 73
- in coronary occlusion, 73
- in pheochromocytoma, 136
- Xhellen, 79
- Mecholyl, 40
- morphine, 92
- Thorazine, 74
- treatment, 74
- with quinidine, 20
- W
- Water, excretion, 63
 intake, 65
- Weakness, in salt depletion, 68
 in syncope, 113
 magnesium sulfate, 46
- Weight, "dry", 68
 reduction in heart failure, 66
- Wheezing, in cardiac asthma, 57
 in pulmonary edema, 58
- Wolff-Parkinson-White, and ventricular tachycardia, 42
- Warming, in coronary occlusion, 75
- X
- X-ray in dissecting aneurysm, 140
 in traumatic heart disease, 144

